

**IN THE U.S. DISTRICT COURT
FOR THE NORTHERN DISTRICT OF OHIO
EASTERN DIVISION**

BRYAN J. PESTA,

Plaintiff,

- against -

CLEVELAND STATE UNIVERSITY BOARD OF
TRUSTEES, in their official capacities, and
LAURA BLOOMBERG, in her individual and official
capacity; and HARLAN M. SANDS, BENJAMIN WARD,
CHRISTOPHER MALLET, CONOR McLENNAN, and
WENDY REGOECZI, in their individual capacities;

Defendants.

Case No. 1:23-cv-00546

JUDGE POLSTER

**AMENDED COMPLAINT
FOR MONEY DAMAGES
AND DECLARATORY AND
INJUNCTIVE RELIEF**

DEMAND FOR JURY ON ALL ISSUES SO TRIABLE

NATURE OF THE ACTION

- 1) This case presents claims based on the Defendants' violation of Dr. Pesta's First Amendment rights to academic freedom, freedom of speech, and freedom of association.
- 2) It involves a flagrant violation of First Amendment rights in the heart of an institution which by its very nature is supposed to be "peculiarly the marketplace of ideas." Instead of that marketplace of ideas, Defendants herein threw a "pall of orthodoxy" over speech which is both controversial and critical of government policy.

PARTIES, JURISDICTION, AND VENUE

- 3) Plaintiff Bryan J. Pesta is a resident of Cuyahoga County, within the Northern District of Ohio.
- 4) The Cleveland State Board of Trustees is the governing body of Cleveland State University and is/are a policy making official(s) for Cleveland State University (CSU).

- 5) The CSU Board of Trustees is currently composed of the natural persons David M. Reynolds, Timothy J. Cosgrove, Patricia M. DePompeii, Paul J. Dolan, Lisa K. Kunkle, Alan G. Sarkoff, Nikki C. Byrd, P. Kelly Tompkins, and Vanessa L. Whiting, who are individuals, and who are also state officials in such capacity. They will be referred to collectively as the “Cleveland State Board of Trustees.” In such official capacity, they / it are sued under Count 3 only.
- 6) Defendant Laura J. Bloomberg was at all times herein the Provost of CSU and as such was the chief academic officer of CSU, although she has now succeeded to the presidency of CSU; moreover, as Provost her recommendations are nearly final decisions, binding on CSU, and rendering her behavior an act or acts of official government policy. She is sued in her individual capacity under Counts 1 and 2; but in her official capacity under Count 3.
- 7) Defendant Harlan M. Sands was at all times herein the President of Cleveland State University and was the chief executive officer of CSU, and as such is a final policy maker in his own right, rendering his behavior an act or acts of official government policy. He is sued in his individual capacity under Count 1 and 2 only.
- 8) Defendant Dr. Benjamin Ward was at all relevant times herein the Director, Research Development & Ethics in CSU’s Office of Research and a participant in a committee investigating Dr. Pesta. He is sued in his individual capacity under Count 1 and 2 only.
- 9) Defendant Dr. Christopher Mallett was at all relevant times herein a Professor of Social Work at CSU and a participant in the committee investigating Dr. Pesta. He is sued in his individual capacity under Count 1 and 2 only.

10) Defendant Dr. Conor McLennan was at all relevant times herein a Professor of Psychology at CSU and a participant in a committee investigating Dr. Pesta. He is sued in his individual capacity under Count 1 and 2 only.

11) Defendant Dr. Wendy Regoeczi was at all relevant times herein the Chairman of, and a Professor in, the Department of Criminology, Anthropology, and Sociology at CSU and a participant in a committee investigating Dr. Pesta. She is sued in her individual capacity under Count 1 and 2.

12) Venue is therefore appropriate in the Northern District of Ohio.

13) This claim involves constitutional rights and is brought pursuant to 42 U.S.C. §1983, as well as 28 USC §§2201-2202.

14) This Court therefore has subject matter jurisdiction pursuant to 28 U.S.C. §1331.

FACTUAL BACKGROUND

15) Dr. Pesta holds a Ph.D. in psychology from the University of Akron; an undergraduate and master's degree in psychology from CSU, and a master's degree in human resource management and labor relations, also from CSU.

16) For over 36 years, from 1986 until March 4, 2022, Pesta was associated with CSU, first as a student, and then later as a faculty member for well over two decades.

17) Dr. Pesta was granted tenure at CSU in 2010, and promotion to full professor in 2016.

18) Indeed, Dr Pesta was, at the time of the events herein, not only a tenured full professor, but he was also the most senior member of his department, with excellent student evaluations.

19) As a psychologist, Dr. Pesta has conducted research and published peer-reviewed papers on the subject of human intelligence (IQ).

20) This area of research is well founded, with IQ in particular being one of the most rigorously investigated metrics in psychology, and with racial differences or gaps in average IQ being well documented for over one hundred years.

21) And yet researching and discussing the cause of the gaps is difficult in part because controversy is generated by even asking questions about this subject.

22) However, in the last 15 years or so the whole-genome revolution (driven by the advent of low-cost DNA testing) has made it possible to do scientifically compelling tests on this topic.

23) Dr. Pesta's research, which resulted in the paper referred to below as “Global Ancestry and Cognitive Ability” (a/k/a “Lasker et al. 2019”), was the first research to be done with a large sample size (over 9,421 subjects) of excellent data provided by the National Institutes of Health.

24) It was the first known study using actual DNA samples (as opposed to measures utilized in the past, such as estimates of probable genetic material inferred from family relationships) while also controlling for the socio-cultural “baggage” of self-identified race.

25) Indeed, “Global Ancestry and Cognitive Ability” (a/k/a “Lasker et al. 2019”) might rightfully be regarded as landmark study for several objective reasons:

- a) First, an entity called “Altmetric (www.altmetric.com)” evaluates articles (across all of science) and ranks them on how impactful they are. The Lasker et al. paper scores in the 99th percentile for “High Attention,” and in the 95th percentile for all research outputs that Altmetric tracks, across disciplines in both “hard” (e.g., chemistry) and social science (e.g., psychology).

- b) Second, the journal that published Lasker et al. (*Psych*) reports that the article had been individually viewed by 53,174 persons to the date of the first complaint in this case. This count represents the most in the journal's history.
- c) Third, Google Scholar reports that the Lasker et al. paper had already been cited 25 times to the date of the first complaint in this case, further illustrating the paper's relative impact and importance.

26) Like all science, Dr. Pesta's research is not the last word on the topic, but it is far more sophisticated and revealing than previous research and should be seen as a landmark of sorts. Instead of being greeted as an academic landmark, Dr. Pesta's research has been the occasion for violations of academic freedom and First Amendment outrages.

27) Pesta's research and publishing herein constitute attempts to communicate his views about a public matter.

28) Furthermore, Pesta's research herein contains premises which challenge those of powerful government policies.

29) On August 30, 2019, Dr. Pesta (along with three other individuals) caused to be published an article entitled "Global Ancestry and Cognitive Ability" (a/k/a "Lasker et al. 2019") in the peer reviewed journal *Psych*. A true and accurate copy of "Global Ancestry and Cognitive Ability" is attached hereto as Exhibit 1.

30) "Global Ancestry and Cognitive Ability" proved to be controversial because it openly included data showing that European American (EA) and African American (AA) populations differ in mean general cognitive ability (or general intelligence, "g") by about one standard

deviation—a fact which has long been established among expert psychometricians, but one which has nevertheless been interdicted by powerful taboos.

31) More controversial still, “Global Ancestry and Cognitive Ability” cited earlier studies showing reasonably strong evidence that genetics played a role in the mean differences in general intelligence between White and Black Americans, and concluded that the data supported those earlier studies, *viz.* Exhibit 1, pdf. p. 23 “4.4. General Conclusion.”

32) These premises conflict directly with those which underly the civil rights policies of the government at all levels, which civil rights policies are based on the premise that the observable differences between Blacks and Whites in general intelligence are due primarily or even exclusively to environmental factors.

33) Yet if Pesta’s research in “Global Ancestry and Cognitive Ability” is accurate, then these differences are in part based on genetics; and men are not only shaped by their environment, but men also, in turn, shape their environment — and they shape it from the powers and deficits bequeathed by their genes.

34) There is thus a direct conflict between the speech of Dr. Pesta and powerful government policies.

35) Yet criticism of government and government policy is not simply a right, but even a duty in our constitutional order, as Justice Brandeis noted long ago.

36) Furthermore, criticism of government actors and government policy is at the very core of the First Amendment, as Justice Brennan noted long ago.

37) Consideration of Dr. Pesta’s point of view is necessary if we would uphold the fundamental First Amendment right of canvassing public men and public measures and

maintaining (in Madison’s words) “the censorial power” of “the people over the Government, and not in the Government over the people.”

38) Open discussion of racial differences in America is confined by powerful taboos which are evidenced by numerous widely reported events going back decades – the very opposite of the “uninhibited, robust, and wide-open debate” our First Amendment supposedly fosters.

39) Upon information and belief, these taboos were known and understood by the hierarchy at CSU at the time of the events herein, including the individuals comprising the Prior Board of Trustees at CSU, President Sands, Provost Bloomberg, and others, including Dr. Benjamin Ward, Dr. Christopher Mallett, Dr. Conor McLennan, and Dr. Wendy Regoeczi.

40) Thus, the controversial nature of openly discussing intelligence differences among the races and the evidence for an innate or genetic explanation for such differences was well known to the above individuals and entities.

41) Turning back to Dr. Pesta’s controversy, in response to “Global Ancestry and Cognitive Ability,” individuals from both inside and outside of the CSU community claimed said article was “racist,” and launched a campaign to have him fired over the said article.

42) Four such individuals who complained to CSU are Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, and Os Keyes.

43) Specifically, on or about September of 2019, Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, and Os Keyes sent a letter to CSU citing Dr. Pesta’s contribution to “Global Ancestry and Cognitive Ability” and complained that the study by Dr. Pesta which resulted in “Global Ancestry and Cognitive Ability” involved “extremely sensitive phenotypic information about ethnicity, cognitive ability, mental health, and educational attainment.”

44) Said letter by Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, and Os Keyes stressed the “extremely sensitive” information as a way of enforcing these taboos, a point which was not lost on the CSU Administration.

45) Furthermore, on or about the Fall/Winter of 2019, in response to “Global Ancestry and Cognitive Ability,” a CSU masters student named Liam O’Brien began to organize other students to petition the CSU administration to censor Dr. Pesta because of the content and viewpoint presented in his research, which agitation bore fruit.

46) Further still, by July 1, 2020, in response to “Global Ancestry and Cognitive Ability,” a student group at CSU known as the “Student Socialist Society CSU” had an ongoing campaign in which they were petitioning the CSU Administration to fire Dr. Pesta on the basis of his speech.

47) Further still, on September 1, 2020, in response to “Global Ancestry and Cognitive Ability,” an individual identified as Theo Desmond – purporting to be with a group known as the “Concerned Citizens Against Race Science” – sent an email to the CSU Department of Management, Provost Jianping Zhu, and the then CSU Board of Trustees, accusing Dr. Pesta of “white nationalism and racism in science.”

48) Said September 1, 2020, email by Theo Desmond attacked the speech and associations of Dr. Pesta and then rhetorically asked that Dr. Pesta be fired with the words, “Is this the type of person the CSU Monte Ahuja School of Business wants its faculty to have professional collaborations with?”

49) Said September 1, 2020, email by Theo Desmond also included the following attack on Dr. Pesta because of his speech and associations:

“Because of this and Pesta's many papers on scientific racism, we demand to know if Pesta is teaching people of color in the classroom. We demand to know if grading is anonymized in the classroom to control for Pesta's biases against students of color. We demand to know if Pesta is following the correct university, state, and federal protocols when sharing data with Kirkegaard and other collaborators.”

50) On September 11, 2020, Dr. Pesta met with CSU's Dean Kenneth Kahn over zoom because of Theo Desmond's demands.

51) Instead of defending Dr. Pesta's First Amendment rights from would-be censors like Theo Desmond, Dean Kahn indicated that the CSU Administration would be subjecting Dr Pesta to an investigation along the lines suggested by Theo Desmond.

52) Upon information and belief, just such an investigation was undertaken at the behest of the CSU Administration in the wake of the meeting on September 11, 2020, which yielded no evidence of any malicious animus on the part of Dr. Pesta.

53) Further still, on or about April 6, 2021, another individual identified as Dr. Kent Taylor, hailing from the Lundquist Institute, and purporting to be a professor of pediatrics at UCLA School of Medicine, wrote CSU's President Sands.

54) In said letter, the individual identified as Dr. Kent Taylor referenced “Global Ancestry and Cognitive Ability,” and stated that “The authors offered the conclusion that, ‘Results converge on genetics as a potential partial explanation for group mean differences in intelligence.’ Use of NIH data for studies of racial differences in this way is both a violation of data use agreement and unethical.”

55) Thus, Dr. Kent Taylor: a) identified a conclusion of Dr. Pesta's work which happens to be at variance with government policy and is interdicted by powerful taboos; and b) instead of squarely disputing the truth or falsity of the conclusion at variance with government policies, accused Dr. Pesta of ethical violations as a means of silencing Dr. Pesta without having to refute his conclusions.

56) The activists succeeded as CSU buckled under the pressure of the controversy generated by the censors identified as Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, Os Keyes, Liam O'Brien, Theo Desmond, Dr. Kent Taylor, as well as the "Student Socialist Society CSU."

57) On or about the Spring of 2021, the CSU Administration, including President Sands, Provost Bloomberg, Dr. Benjamin Ward, and others instituted a committee to investigate Dr. Pesta because of his part in "Global Ancestry and Cognitive Ability."

58) In taking such action, the CSU Administration, including President Sands, Provost Bloomberg, Dr. Benjamin Ward, and others, were acting at the behest of the censors identified as Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, Os Keyes, Liam O'Brien, Theo Desmond, Dr. Kent Taylor, as well as the "Student Socialist Society CSU," all of whom had taken issue with the content and viewpoint of Dr. Pesta's speech.

59) The CSU Administration, including President Sands, Provost Bloomberg, Dr. Benjamin Ward, and others, did not take any adverse actions against any CSU students or faculty who were critical of Dr. Pesta's speech, such as Liam O'Brien, or any students or faculty connected to the "Student Socialist Society at CSU."

60) However, in taking such action against Dr. Pesta, the CSU Administration engaged in viewpoint discrimination.

61) In taking such action, the CSU Administration, including President Sands, Provost Bloomberg, Dr. Benjamin Ward, and others, all knew they were working to silence a controversial and unpopular point of view.

62) The committee so instituted consisted of Dr. Christopher Mallett, Dr. Conor McLennan, and Dr. Wendy Regoeczi, with the cooperation of Dr. Benjamin Ward, and others.

63) The committee members investigating Dr. Pesta accepted their appointments on or about June 29, 2021.

64) Prior to conducting any of the interviews related below, the committee members all read through and familiarized themselves with the September of 2019 joint letter from Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, and Os Keyes, as well as the April 6, 2021, letter from Dr. Kent Taylor.

65) So too did Provost Bloomberg read through and familiarize herself with the September of 2019 joint letter from Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, and Os Keyes, as well as the April 6, 2021, letter from Dr. Kent Taylor.

66) So too did President Sands read through and familiarize himself with the September of 2019 joint letter from Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, and Os Keyes, as well as the April 6, 2021, letter from Dr. Kent Taylor.

67) Thus, even before any interviews were undertaken, the committee of Dr. Christopher Mallett, Dr. Conor McLennan, and Dr. Wendy Regoeczi, along with Dr. Benjamin Ward, and Provost Bloomberg, and President Sands all understood that content-specific and viewpoint specific considerations loomed heavily over the investigation that was being undertaken.

68) On September 7, 2021, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward, conducted an interview with Dr. Pesta.

69) In the course of said interview, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward specifically acknowledged that Pesta was working and publishing in a controversial field.

70) As such, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward, all had notice that Pesta would need the maximum protections offered by academic freedom.

71) On September 7, 2021, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward, were informed by Pesta that he had already been intimidated to such an extent that he was willing to retract his controversial research, regardless of the fact that it had not been squarely disputed.

72) Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward were therefore on notice that they were failing to effectively secure Pesta's academic freedom.

73) Indeed, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward, understood that they themselves had effected the violation of Pesta's academic freedom and First Amendment rights.

74) Indeed, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward, understood that their actions could result in the diminution of knowledge and would contribute to ignorance.

75) Instead of reversing course, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward, continued on an unlawful path with the conscious intent to foment ignorance.

76) On September 30, 2021, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward interviewed Dr. Jedidiah Carlson, Dr. Cathryn Townsend, and Kevin Bird, who had previously complained about Pesta.

77) In the course of said interview, Dr. Jedidiah Carlson, Dr. Cathryn Townsend, and Kevin Bird again made clear that their chief complaint against Pesta lay with his conclusions on the genetic basis for the mean differences in general intelligence between White and Black Americans.

78) Thus, in the course of said interview, Dr. Jedidiah Carlson, Dr. Cathryn Townsend, and Kevin Bird made statements such as “I pay attention to racist applications of genetic data and groups that do publish research aligning one way, especially the many of the co-authors on this paper [*viz.* Pesta’s “Global Ancestry and Cognitive Ability” a/k/a “Lasker et al. 2019”].”

79) Indeed, Dr. Jedidiah Carlson, Dr. Cathryn Townsend, and Kevin Bird made clear that they only began looking into the methodology of Pesta’s research because they were hostile to the conclusions Pesta had reached, stating “And so we started looking into, [*sic*] was this actually inappropriate use of NIH data? Are there, or was there, something kind of gone wrong when it was used for a paper like this?”

80) Kevin Bird in particular took issue with what he said was “[testing] basically the genetic hypothesis of the cause of the racial IQ gap.”

81) Indeed, on September 30, 2021, Kevin Bird and his colleagues repeatedly acknowledged that there is in fact a “racial IQ gap,” but made clear that what they took issue with was any probing of the point of view that such “racial IQ gap” was caused by genetic as opposed to environmental factors, *e.g.*:

“something like the hereditarian hypothesis, kind of a long-standing racist theory, would probably not be within their purview of ethical research.”

And:

“[T]here would need to have been some kind of justification for why the research provided some kind of benefit that outweighed any potential risk to this research, which I don’t really see. I don’t see any kind of plausible argument that they could have made for that, if they had been truthful about the purpose.”

82) Kevin Bird and colleagues made clear that they wanted no testing of “the hereditarian hypothesis” of “the racial IQ gap,” meaning that they expected CSU to throw a “pall of orthodoxy” over this subject.

83) On September 28, 2021, Dr. Wendy Regoeczi sent an email to another person who had complained about Pesta, Dr. Kent Taylor; specifically, Dr. Wendy Regoeczi contacted Dr. Taylor in anticipation of an interview that would be held on October 5, 2021.

84) In said email of September 28, 2021, Dr. Wendy Regoeczi, asked Dr. Taylor to expand on his earlier letter to President Sands and explain how Pesta’s use of NIH data for studies of racial differences was “both a violation of the data use agreement and unethical.”

85) On September 29, 2021, Dr. Taylor responded with an email of his own.

86) In said response, the very first thing Dr. Taylor mentioned was his concern with the conclusion of “Global Ancestry and Cognitive Ability” (a/k/a “Lasker et al., 2019”), viz. “...my concern is with the final sentence of the abstract of Lasker et al., 2019, ‘Results converge on genetics as a potential partial explanation for group mean differences in intelligence.’ In my opinion, this statement conflicts with the NIH policy NOT-OD-07-088 on taking care that data avoids stigmatization of US population sub-groups.”

87) Dr. Taylor went on: “In my opinion use of dbGaP data to support the conclusion that there are genetic differences in intelligence is an unethical use of the data that my colleagues and I work so hard to collect to advance medial science. I do recognize that Dr. Pesta disagrees with me on this issue, well documented by the various articles published in psych[sic] ...”

88) Moreover, during Dr. Taylor’s October 5, 2021, interview with Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward, Dr. Taylor made clear that he was firmly in the “environment only” camp that is ideologically opposed to Dr. Pesta’s point of view, *e.g.*:

“... there’s lots of things missing [from “Global Ancestry and Cognitive Ability”]. And there’s lots of things that would address the issue. [sic] There’s a lot of things on socio-economic status that are missing, there’s a lot of considerations that are most likely in the data set that are not here in this paper.”

89) Also, during the interview of Dr. Taylor on October 05, 2021, committee members asked:

“Would an [Institutional Review Board] be required to approve [the ethics of Pesta’s research]?”

To which Dr. Taylor replied:

“I’m sorry, I don’t know that an IRB would approve it...[Lasker et al.] is stigmatization, and we’re very careful not to do this sort of thing, quite frankly, it’s a misuse of the data. We have so many research questions – why are we doing this?”

Thereafter, at that same meeting, committee members asked Dr. Taylor:

“I’m wondering in your professional opinion if you think the work from Dr. Pesta and his colleagues deviates from commonly accepted practice in the academic community for proposing, conducting, and reporting research?”

To which Dr. Taylor replied:

“In my work, this is clearly outside of what you should be doing.”

90) In both his email and his Zoom interview, Dr. Taylor was therefore confirming that what alarmed him most was the viewpoint expressed in Pesta’s conclusion about the link between genetics, race, and intelligence.

91) Indeed, on October 5, 2021, Dr. Taylor made clear that his intent was to interdict any inquiry into the subject of genetics, race, and intelligence, flatly stating:

“The reason I wrote to the President was it was just concerning that this is not something that could be done... [sic]... We have cognitive data as well; I could not use it for this kind of a study in MESA or TOPMed. [sic] Any study that I know. But I did notice that we (MESA, TOPMed) specifically say it can’t be used for this in the agreements as part of the dbGaP documentation... That it can’t be used for this kind of study of the subpopulations in the county.”

92) In the above statement, Dr. Taylor betrayed the fact that even in the hard sciences there are powerful constraints against “uninhibited, robust and wide-open debate” where the subject is genetics, race, and intelligence.

93) Furthermore, in the above statement Dr. Taylor made clear that he expected those constraints to take hold even in the academy, where he and those of his ideological persuasion would throw a “pall of orthodoxy” over this subject and prohibit inquiry.

94) Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward understood that Dr. Taylor was demanding a “pall of orthodoxy.”

95) Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward understood that Dr. Taylor’s pall of orthodoxy was a flagrant violation of academic freedom and Dr. Pesta’s First Amendment rights.

96) Nevertheless, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward intentionally violated Dr. Pesta’s academic freedom and First Amendment rights by acceding to Dr. Taylor’s unlawful and totalitarian demands.

97) The committee would go on to issue a report dated January 13, 2022, which stated, “The Committee members were, of course, aware of the research topics investigated and the controversial nature of the scholarship in which Dr. Pesta and his colleagues examined race/ethnicity, heritable traits, and intellectual ability; a controversy with a long history and a more recent undercurrents,” which again serves to underscore the fact that the committee knew of the controversial nature of Dr. Pesta’s work when taking action against him.

98) The said committee of Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward recommended terminating Dr. Pesta.

99) The said committee of Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward recommended terminating Dr. Pesta's as a way to appease censors such as Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, Os Keyes, Liam O'Brien, Theo Desmond, Dr. Kent Taylor, as well as the "Student Socialist Society CSU," or for other illicit reasons.

100) The said committee of Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward thereby undertook viewpoint discrimination against Dr. Pesta.

101) The said committee of Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward knowingly and intentionally undertook such invidious viewpoint discrimination against Dr. Pesta.

102) Indeed, throughout the interviews on both September 30, 2021, and October 5, 2021, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward signaled their intent to placate Dr. Pesta's critics by failing to ever defend his rights; and even going so far as to plead with, and fawn over, the censors calling for viewpoint discrimination against Dr. Pesta, *e.g.* in response to the clear calls for viewpoint discrimination on September 30, 2021, Dr. Christopher Mallett assured Pesta's censors that "Hopefully, you can see that we, as a university take this very seriously..."

103) One of the ways, amongst others, that the CSU administration, including President Sands and Provost Bloomberg, signaled its intent to appease Dr. Pesta's critics was by removing links to prior work by Pesta on its "Engaged Scholarship at CSU" page.

104) CSU maintains a webpage exhibiting the socially relevant scholarship of scholars affiliated with CSU at a web page found at <https://engagedscholarship.csuohio.edu/>.

105) Prior to the complaints of would be censors Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, Os Keyes, Liam O'Brien, Theo Desmond, Dr. Kent Taylor, as well as the "Student Socialist Society CSU," CSU had maintained a link to another article Pesta had authored that dealt with the Black-White IQ gap.

106) That earlier article was entitled "Black-White differences on IQ and grades: The mediating role of elementary cognitive tasks" which Pesta had published in 2008, in the elite academic journal *Intelligence* (*viz. Intelligence*, 36, 323-329).

107) However, in the wake of the controversy over "Global Ancestry and Cognitive Ability," CSU removed the link to Dr. Pesta's "Black-White differences on IQ and grades: The mediating role of elementary cognitive tasks" article from its "Engaged Scholarship at CSU" page.

108) Nor has CSU ever linked to "Global Ancestry and Cognitive Ability" on its "Engaged Scholarship at CSU" page, which is another manifestation of CSU's censorious hostility to Dr. Pesta and its intent to maintain the unlawful palls of orthodoxy demanded by censors such as Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, Os Keyes, Liam O'Brien, Theo Desmond, Dr. Kent Taylor, as well as the "Student Socialist Society CSU."

109) By decision dated January 13, 2022, which was effected March 4, 2022, Provost Bloomberg accepted the recommendation of the said committee and terminated Dr. Pesta's employment at CSU.

110) In taking such action, Provost Bloomberg also acted to appease the censors identified as Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, Os Keyes, Liam O'Brien, Theo Desmond, Dr. Kent Taylor, as well as the "Student Socialist Society CSU," or for other illicit reasons.

111) In taking such action, Provost Bloomberg had monitored and then reviewed the investigation of Dr. Pesta by Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoecki, and Dr. Benjamin Ward, including the interviews on September 7, 2021, September 30, 2021, October 5, 2021, and October 11, 2021.

112) In taking such action, Provost Bloomberg knew that she was taking part in viewpoint discrimination, which she understood was an egregious First Amendment violation.

113) President Sands was aware of the actions taken against Dr. Pesta, as he had been monitoring Dr. Pesta's situation at least since October 7, 2020, when he received two letters from the Foundation for Individual Rights and Expression (FIRE) sent on behalf of Dr. Pesta, which defended Pesta's First Amendment rights with respect to the controversy being instigated by the likes of Pesta's would be censors over "Global Ancestry and Cognitive Ability."

114) At any time throughout, President Sands, as the chief executive officer of CSU, held authority over Provost Bloomberg, and the investigative committee of Dr. Pesta by Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoecki, and Dr. Benjamin Ward, and could have halted or overruled their actions.

115) However, President Sands either acquiesced in the actions taken against Dr. Pesta or even countenanced such actions.

116) President Sands, Provost Bloomberg, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoecki, and Dr. Benjamin Ward were clothed with authority from the state and acted under color of law and were state actors.

117) Nevertheless, as state actors operating under color of law, President Sands, Provost Bloomberg, Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoecki, and Dr.

Benjamin Ward came into conflict with the superior authority of the Constitution, the First and Fourteenth Amendments, and were therefore stripped of their official or representative character and are rightly subjected in their persons to the consequences of their lawless conduct.

COUNT I: FIRST AMENDMENT RETALIATION AGAINST PROVOST BLOOMBERG, PRESIDENT SANDS, AND PROFESSORS MALLETT, McLENNAN, REGOECZI, AND WARD FOR THE INVESTIGATION OF DR. PESTA.

118) Dr. Pesta restates the allegations of Paragraphs 1 through 117 and incorporates them here as if fully rewritten.

119) Dr. Pesta spoke as a citizen and not as an employee in his contributions to “Global Ancestry and Cognitive Ability.”

120) In the alternative, it is irrelevant whether Dr. Pesta was speaking as a citizen and not an employee by his contributions to “Global Ancestry and Cognitive Ability.”

121) Dr. Pesta’s speech in “Global Ancestry and Cognitive Ability” involved matters of public concern.

122) Provost Bloomberg, President Sands, and Professors Mallett, McLennan, Regoeczi, and Ward lacked any justification for treating Dr. Pesta differently from the general public based on CSU’s needs as an employer under any balancing tests.

123) Indeed, on the contrary, said Defendants owed an affirmative duty to Dr. Pesta to provide an atmosphere of free speech where controversial positions could be probed without regard to offending the likes of Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, Os Keyes, Liam

O'Brien, Theo Desmond, Dr. Kent Taylor, the "Student Socialist Society CSU, or any other person or entity offended by "Global Ancestry and Cognitive Ability."

124) Dr. Pesta therefore engaged in protected speech when he contributed to "Global Ancestry and Cognitive Ability."

125) Provost Bloomberg, President Sands, and Professors Mallett, McLennan, Regoeczi, and Ward took adverse action against Dr. Pesta that would deter a person of ordinary firmness from continuing to engage in such conduct when they caused an investigation and investigated Dr. Pesta.

126) Furthermore, President Sands acquiesced in or even countenanced Dr. Pesta's investigation.

127) That investigation was motivated at least in part by Dr. Pesta's protected conduct in contributing to "Global Ancestry and Cognitive Ability."

128) As a proximate result, Dr. Pesta was injured in his good name, vocation, and well-being; and is entitled to damages from Defendants.

129) The individual Defendants Provost Bloomberg, President Sands, and Professors Mallett, McLennan, Regoeczi, and Ward are all academics of decades long standing within the academy and as such fully understood the importance of freedom of speech and academic freedom for the proper functioning of a state university.

130) In taking such actions, the above individual Defendants were motivated by conscious ill will or malice, or at the very least, a reckless disregard of Dr. Pesta's rights, warranting punitive damages.

131) Furthermore, the above individual Defendants were conscious of the fact that they were acceding to viewpoint discrimination, one of the most egregious forms of First Amendment violations, which warrants punitive damages.

COUNT II: FIRST AMENDMENT RETALIATION AGAINST PROVOST BLOOMBERG, PRESIDENT SANDS, AND PROFESSORS MALLETT, McLENNAN, REGOECZI, AND WARD FOR FOR THE TERMINATION OF DR. PESTA.

132) Dr. Pesta restates the allegations of Paragraphs 1 through 131 and incorporates them here as if fully rewritten.

133) Dr. Pesta spoke as a citizen and not as an employee in his contributions to “Global Ancestry and Cognitive Ability.”

134) In the alternative, it is irrelevant whether Dr. Pesta was speaking as a citizen and not an employee by his contributions to “Global Ancestry and Cognitive Ability.”

135) Dr. Pesta’s speech in “Global Ancestry and Cognitive Ability” involved matters of public concern.

136) Provost Bloomberg, President Sands, and Professors Mallett, McLennan, Regoeczi, and Ward lacked any justification for treating Dr. Pesta differently from the general public based on CSU’s needs as an employer under any balancing tests.

137) Indeed, on the contrary, said Defendants had an affirmative duty to Dr. Pesta to provide an atmosphere of free speech where controversial positions could be probed without regard to offending the likes of Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, Os Keyes, Liam

O'Brien, Theo Desmond, Dr. Kent Taylor, the "Student Socialist Society CSU, or any other person or entity offended by "Global Ancestry and Cognitive Ability."

138) Dr. Pesta therefore engaged in protected speech when he contributed to "Global Ancestry and Cognitive Ability."

139) Provost Bloomberg and President Sands took adverse action against Dr. Pesta that would deter a person of ordinary firmness from continuing to engage in such conduct when they fired Dr. Pesta, based on the report issued by Professors Mallett, McLennan, Regoeczi, and Ward, by decision dated January 13, 2022, which was effective March 4, 2022.

140) Furthermore, President Sands acquiesced in or even countenanced Dr. Pesta's firing.

141) That firing was motivated at least in part by Dr. Pesta's protected conduct in contributing to "Global Ancestry and Cognitive Ability."

142) As a proximate result, Dr. Pesta was injured in his good name, vocation, and well-being; and is entitled to damages from Defendants, including but not limited to both back pay and front pay.

143) The individual Defendants Provost Bloomberg, President Sands, and Professors Mallett, McLennan, Regoeczi, and Ward are all academics of decades long standing within the academy and as such fully understood the importance of freedom of speech and academic freedom for the proper functioning of a state university.

144) In taking such actions, the above individual Defendants were motivated by conscious ill will or malice, or at the very least, a reckless disregard of Dr. Pesta's rights, warranting punitive damages.

145) Furthermore, the above individual Defendants were conscious of the fact that they were acceding to viewpoint discrimination, one of the most egregious forms of First Amendment violations, which warrants punitive damages.

**COUNT III: DECLARATORY AND INJUNCTIVE RELIEF AGAINST
DEFENDANTS CLEVELAND STATE UNIVERSITY BOARD OF TRUSTEES
AND BLOOMBURG IN THEIR OFFICIAL CAPACITIES.**

146) Dr. Pesta restates the allegations of Paragraphs 1 through 145 and incorporates them here as if fully rewritten.

147) Defendants Cleveland State Board of Trustees (by which Pesta means the individual state officials David M. Reynolds, Timothy J. Cosgrove, Patricia M. DePompeii, Paul J. Dolan, Lisa K. Kunkle, Alan G. Sarkoff, Nikki C. Byrd, P. Kelly Tompkins, and Vanessa L. Whiting in their official capacities) and Bloomberg are policymaking officials for CSU.

148) There is presently at CSU a *de facto* policy of refusing to extend academic freedom to those such as Dr. Pesta who research and publish on the controversial topic of race, genes, and intelligence from the hereditarian point of view.

149) The policy is implemented and enforced at the direction of Defendant Bloomberg with the knowledge, consent, or acquiescence of the Cleveland State Board of Trustees.

150) Indeed, most of the events surrounding Pesta's investigation and termination, relayed above, took place under the watch of the "Prior Board of Trustees," which consisted of David M. Reynolds, Thomas W. Adler, Timothy J. Cosgrove, Patricia M. DePompeii, Paul J. Dolan, Stephen F. Kirk, Lisa K. Kunkle, Alan G. Starkoff, and Vanessa L. Whiting.

151) Comparing the membership of the Prior Board of Trustees with the Defendants Cleveland State Board of Trustees reveals that only Nikki C. Byrd and P. Kelly Tompkins are new additions (replacing Thomas W. Adler and Stephen F. Kirk).

152) Upon information and belief, the Prior Board of Trustees had also monitored Dr. Pesta's situation at least since the institution of the investigative committee comprised of Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward, and could have halted or overruled their actions.

153) Upon information and belief, the Prior Board of Trustees had specifically considered the investigation of Dr. Pesta during executive sessions of the Board of Trustees on July 26, 2021, September 23, 2021, October 27, 2021, November 18, 2021, January 27, 2022, and March 17, 2022.

154) Upon information and belief, at some of all of these meetings, the Prior Board of Trustees were briefed by President Sands, or Provost Bloomberg, or both, as to the actions being taken against Dr. Pesta.

155) Upon information and belief, the Prior Board of Trustees had monitored and then reviewed the investigation of Dr. Pesta by Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward, including the interviews on September 7, 2021, September 30, 2021, October 5, 2021, and October 11, 2021, as well as the September of 2019 joint letter from Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, and Os Keyes, as well as the April 6, 2021, letter from Dr. Kent Taylor.

156) Thus, the Prior Board of Trustees understood that content-specific and viewpoint specific considerations loomed heavily over the investigation of Dr. Pesta.

157) However, the Prior Board of Trustees also either acquiesced in the actions taken against Dr. Pesta or even countenanced such actions.

158) The Prior Board of Trustees officially approved of Defendant Bloomberg's termination of Dr. Pesta's employment at CSU effective March 4, 2022.

159) Upon information and belief, a majority of the Prior Board of Trustees did so at an executive session of the Board of Trustees on January 27, 2022.

160) In taking such action, the Prior Board of Trustees also acted to appease the censors identified as Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, Os Keyes, Liam O'Brien, Theo Desmond, Dr. Kent Taylor, as well as the "Student Socialist Society CSU," or for other illicit reasons.

161) But as stated above, most of the individual member of the Prior Board of Trustees have continued on as the present Defendants Cleveland State Board of Trustees and continue to acquiesce in or even countenance the *de facto* policy at CSU of refusing to extend academic freedom to those such as Dr. Pesta who research and publish on the controversial topic of race, genes, and intelligence from the hereditarian point of view.

162) This *de facto* policy has directly harmed Pesta through the investigation and termination detailed above, as well as in the refusal of CSU to link to Pesta's research on its "Engaged Scholarship at CSU" page and the continued disruption of Pesta's tenure.

163) Accordingly, there is an actual controversy between Pesta and Defendants Cleveland State Board of Trustees and Bloomberg as state officials in their official capacities.

164) Scholarship and education cannot survive in an atmosphere of suspicion.

165) Yet such an atmosphere hangs over any, like Pesta, who advances the “genetic hypothesis of the cause of the racial IQ gap” or the “hereditarian hypothesis” (*viz.* Pesta’s censor, Kevin Bird, at Paragraphs 80 and 81 above).

166) Moreover, in Pesta’s case, the anti-intellectual forces have progressed (or declined) from an atmosphere of suspicion to outright persecution, which now holds sway at CSU, especially under Defendant Bloomberg as President, backed by Defendant Cleveland State Board of Trustees.

167) What is at stake is not simply Pesta’s point of view, but the viability of all scholarship and education in this field at CSU, and likely elsewhere.

168) “Freedom of speech has become a central concern of the Western society because of the discovery among the Greeks that **dialectic, as demonstrated in the Socratic dialogues, is a principal method of attaining truth...** ‘The ability to raise searching difficulties on both sides of a subject will,’ said Aristotle, ‘make us detect more easily the truth and error about the several points that arise.’” – all of which is true.

169) But yet: “The method of dialectics is to confront ideas with opposing ideas in order that the pro and con of the dispute will lead to true ideas.” – which is also true.

170) To the contrary, the demand of censors such as Kevin Bird and his ilk is that the controversial topic of race, genes, and intelligence be spared the rigors of dialectic, so that the “environmental hypothesis” cannot be challenged.

171) Under such intellectual and moral cowardice, even Bird’s “environmental hypothesis” is reduced to a dead dogma, bereft of any grounding in real scholarship or education which has been spared the rigors of dialectic.

172) To be sure, Pesta does not maintain that his own research on this controversial topic is the last or definitive word on the topic; nor does he seek a dogmatic declaration that his research has been proved correct.

173) But Pesta does seek a declaration that such research into the controversial topic of race, genes, and intelligence is under assault for reasons wholly removed from valid scientific criteria, as demonstrated by the statements that CSU itself collected from Dr. Jedidiah Carlson, Dr. Cathryn Townsend, Kevin Bird, and Dr. Kent Taylor which are quoted at Paragraphs 78—81, 86—88, and 91 above.

174) Moreover, this anti-intellectual assault is connected to, and draws strength from, broader taboos within American culture.

175) There is thus the danger that even within the academy, which “occupies a special niche in our constitutional tradition,” a “pall of orthodoxy” will continue to be thrown over this important topic.

176) By such declaration, Pesta’s case would be transformed from an extremely negative example of exemplary destruction, meant to effectively chill academic freedom, to a ringing endorsement of a professor’s right to “to examine all options, no matter how unpopular or unorthodox, without concern that [his] career will be indelibly marred by daring to think along nonconformist pathways.”

177) Accordingly, Pesta seeks a declaratory judgment:

- a) Declaring that the hereditarian hypothesis into the long-standing racial gap in IQ is worthy of study, but is presently under assault for reasons wholly removed from valid scientific criteria; and

- b) Declaring that to those, like Pesta, who advance a hereditarian hypothesis in the study of race, genetics, and intelligence are entitled to academic freedom; and
- c) Declaring that Defendants Cleveland State Board of Trustees and Bloomberg, in particular, must extend full academic freedom to any, such as Pesta, who advance a hereditarian hypothesis in the study of race, genetics, and intelligence; and
- d) Declaring that Defendants Cleveland State Board of Trustees and Bloomberg may not utilize any aspect of the investigation undertaken by Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward to interfere with Pesta's academic freedom.

178) Furthermore, Pesta seeks, and is entitled to, a preliminary injunction prior to judgment in this case and a permanent injunction thereafter enjoining Defendants Cleveland State Board of Trustees and Bloomberg to:

- a) to re-establish the link to Pesta's earlier article entitled "Black-White differences on IQ and grades" on the CSU "Engaged Scholarship at CSU" page; and
- b) to establish a link to Pesta's groundbreaking article "Global Ancestry and Cognitive Ability" on the "Engaged Scholarship at CSU" page; and
- c) to extend academic freedom to those, like Pesta, who advance a hereditarian hypothesis in the study of race, genetics, and intelligence; and,
- d) to award Pesta full back pay from the date of termination to the date his pay becomes re-instated; and
- e) to pay Pesta's full salary during the pending of this lawsuit; and

f) from relying on any part of the investigation of Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward to justify terminating Pesta's tenured professorship; and

g) to reinstate Pesta to his full tenured professorship with no further interference of his academic freedom and First Amendment rights based on the investigation of Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward.

179) Pesta will suffer irreparable harm absent such injunctive relief, given that the denial of his regular salary is causing and will continue to cause hardship, which cannot be undone.

180) Pesta is likely to prevail on the merits in this litigation.

181) The balance of equities favors Pesta.

182) The public interest favors Pesta as the public interest itself lies squarely with the vigorous exercise of First Amendment rights and the protection of academic freedom.

WHEREFORE, Dr. Pesta demands:

As to Count One, compensatory damages in an amount to be determined at trial, but in excess of \$25,000, together with punitive and exemplary damages, and attorneys' fees as provided by 42 U.S.C. §§1983 and 1988;

As to Count Two, compensatory damages in an amount to be determined at trial, but in excess of \$25,000, together with punitive and exemplary damages, and attorneys' fees as provided by 42 U.S.C. §§1983 and 1988;

As to Count Three, a declaratory judgment:

- a) Declaring that the hereditarian hypothesis into the long-standing racial gap in IQ is worthy of study, but is presently under assault for reasons wholly removed from valid scientific criteria; and
- b) Declaring that to those, like Pesta, who advance a hereditarian hypothesis in the study of race, genetics, and intelligence are entitled to academic freedom; and
- c) Declaring that Defendants Cleveland State Board of Trustees and Bloomberg in particular must extend full academic freedom to any, such as Pesta, who advance a hereditarian hypothesis in the study of race, genetics, and intelligence; and
- d) Declaring that Defendants Cleveland State Board of Trustees and Bloomberg may not utilize any aspect of the investigation undertaken by Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward to interfere with Pesta's academic freedom.

And a Permanent Injunction enjoining Defendants Cleveland State Board of Trustees and Bloomberg:

- a) to re-establish the link to Pesta's earlier article entitled "Black-White differences on IQ and grades" on the CSU "Engaged Scholarship at CSU" page; and
- b) to establish a link to Pesta's groundbreaking article "Global Ancestry and Cognitive Ability" on the "Engaged Scholarship at CSU" page; and
- c) to extend academic freedom to those, like Pesta, who advance a hereditarian hypothesis in the study of race, genetics, and intelligence; and,

- d) to award Pesta full back pay from the date of termination to the date his pay become re-instated; and
 - e) to pay Pesta's full salary during the pending of this lawsuit; and
 - f) from relying on any part of the investigation of Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward justify terminating Pesta's tenured professorship; and
 - g) to reinstate Pesta to his full tenured professorship with no further interference of his academic freedom and First Amendment rights based on the investigation of Dr. Christopher Mallett, Dr. Conor McLennan, Dr. Wendy Regoeczi, and Dr. Benjamin Ward;
- together with attorney's fee pursuant to 42 U.S.C §§1983 and 1988.

Dated: Goshen, New York
September 22, 2023

Respectfully submitted,

Frederick C. Kelly, Esq.
Law Office of Frederick C. Kelly
Attorney for Plaintiff Bryan J. Pesta
One Harriman Square
Goshen, New York 10924
(845) 294-7945
fckellylaw@protonmail.com
Bar No. NY4377297

EXHIBIT 1



Article

Global Ancestry and Cognitive Ability

Jordan Lasker ¹, Bryan J. Pesta ^{2,*}, John G. R. Fuerst ³ and Emil O. W. Kirkegaard ⁴

¹ Department of Economics, University of Minnesota Twin Cities, Minneapolis, MN 55455, USA

² Department of Management, Cleveland State University, Cleveland, OH 44115, USA

³ Cleveland State University, Cleveland, OH 44115, USA

⁴ Ulster Institute for Social Research, London NW26 9LQ, UK

* Correspondence: b.pest@csuohio.edu

Received: 8 June 2019; Accepted: 28 August 2019; Published: 30 August 2019



Abstract: Using data from the Philadelphia Neurodevelopmental Cohort, we examined whether European ancestry predicted cognitive ability over and above both parental socioeconomic status (SES) and measures of eye, hair, and skin color. First, using multi-group confirmatory factor analysis, we verified that strict factorial invariance held between self-identified African and European-Americans. The differences between these groups, which were equivalent to 14.72 IQ points, were primarily (75.59%) due to difference in general cognitive ability (g), consistent with Spearman's hypothesis. We found a relationship between European admixture and g . This relationship existed in samples of (a) self-identified monoracial African-Americans ($B = 0.78$, $n = 2,179$), (b) monoracial African and biracial African-European-Americans, with controls added for self-identified biracial status ($B = 0.85$, $n = 2407$), and (c) combined European, African-European, and African-American participants, with controls for self-identified race/ethnicity ($B = 0.75$, $N = 7,273$). Controlling for parental SES modestly attenuated these relationships whereas controlling for measures of skin, hair, and eye color did not. Next, we validated four sets of polygenic scores for educational attainment (eduPGS). MTAG, the multi-trait analysis of genome-wide association study (GWAS) eduPGS (based on 8442 overlapping variants) predicted g in both the monoracial African-American ($r = 0.111$, $n = 2179$, $p < 0.001$), and the European-American ($r = 0.227$, $n = 4914$, $p < 0.001$) subsamples. We also found large race differences for the means of eduPGS ($d = 1.89$). Using the ancestry-adjusted association between MTAG eduPGS and g from the monoracial African-American sample as an estimate of the transracially unbiased validity of eduPGS ($B = 0.124$), the results suggest that as much as 20%–25% of the race difference in g can be naïvely explained by known cognitive ability-related variants. Moreover, path analysis showed that the eduPGS substantially mediated associations between cognitive ability and European ancestry in the African-American sample. Subtest differences, together with the effects of both ancestry and eduPGS, had near-identity with subtest g -loadings. This finding confirmed a Jensen effect acting on ancestry-related differences. Finally, we confirmed measurement invariance along the full range of European ancestry in the combined sample using local structural equation modeling. Results converge on genetics as a potential partial explanation for group mean differences in intelligence.

Keywords: race; intelligence; ancestry; socioeconomic status; discrimination; nature/nurture

1. Introduction

The European-American (EA) and African-American (AA) populations have been found to differ in mean general cognitive ability (or general intelligence, g) by about one standard deviation [1–3]. This difference exists even though the heritability of intelligence is both high and virtually identical in the European and African-American populations [4]. Likewise, the shared and unshared environmental components of variance in g are similar in these groups. These findings, in conjunction with others

(see, e.g., [2,5]), have led some to propose the existence of a non-trivial genetic influence on group differences in cognitive ability (e.g., [2,6–9]).

Researchers have devised various methods to assess the causality behind these differences [10,11]. Examples include transracial adoption studies [12,13] (for a review, see [14,15]), analysis of differential regression to the mean [7,16,17], investigations of Spearman's hypothesis [18–20], biometric decomposition of mean group differences [2,21], studies of multiracial persons [11,22], and various intra- and cross-national comparisons [8].

Some researchers have proposed the use of admixture analysis to assess the relationship between genetic ancestry and cognitive ability [10,23–25]. The premise of this approach is that among admixed population's individuals possessing different proportions of genetic ancestry from different populations will be more or less similar to members of these populations if they derive a greater or lesser proportion of their genetic ancestry from them. This simple genetic model predicts that individual resemblance to a given group mean in various outcomes (e.g., height, appearance, intelligence) will be a function of that individual's ancestry derived from that group [24,26]. When a difference in the level of some trait is observed between groups, an admixture analysis can provide indirect evidence that genetics plays a role in the difference.

A number of older studies have attempted to assess the relationship between ancestry and cognitive ability in admixed populations. Most of these studies, however, used indirect measures of ancestry, such as self-reports, genealogical records, or indices based on physical features like hair texture or skin color. These measures do not constitute objective or reliable indices of ancestry. As such, they show substantial measurement error and may thus be prone to giving inaccurate results. This may be especially true when the measures are based on phenotypic indicators of ancestry, as these are confounded with potentially biasing variables like discrimination, or cross-assortative mating for specific traits [1,2,27].

The above problem is common to all classical admixture studies. In addition to uncertainty regarding the extent of admixture, these studies also tend to suffer from small sample sizes, lack of replication, distinctly unrepresentative sampling, and young age at assessment [28]. The issue of age at assessment can be severe, as one of the most well-replicated findings about the behavior genetics of intelligence is that heritability tends to increase with age (this has been dubbed the Wilson effect; [29]). As heritability rises, the shared environmental component of traits like intelligence tends to become insignificant [30,31]. If individuals from different races are reared in similar environments, typically observed differences in cognitive ability may not emerge until adulthood due to these shared environments masking them in childhood.

Modern DNA-based methods of assessing admixture are available, and with them researchers can generate accurate point estimates of individual ancestry from many different groups. Genomic admixture analysis has already proven to be a useful technique for investigating the causes of group differences in a variety of traits. For example, Becker et al. (2011) [32] found that among Central African Pygmies, non-Pygmy admixture was associated with greater height. After the initial association between admixture and a trait is found to be robust, other methods like local admixture mapping are available to determine the specific genes and mechanisms behind group differences. Several authors (see, e.g., [33–36]) have applied these techniques to Pygmies, finding that the Pygmy/non-Pygmy stature gap can be traced in part to specific differences in immune and neuroendocrine functioning.

Admixture analysis has been applied to many other traits, including diabetes [37–39], hypertension [40], adaptation to high altitudes [41], asthma [42], sleep depth [43], risk of prostate cancer [44–47], and uterine fibroids [48]. These studies generally find that admixture relates to the trait under investigation above and beyond covariates. Sometimes, however, the inclusion of putative environmental covariates, like diet or socioeconomic status (SES), removes the association with ancestry (e.g., [49]). This suggests either that environmental covariates mediate the associations between ancestry and outcomes or that ancestry is a non-causal correlate of the trait in question that's actually caused by something else. This suggests either that environmental covariates mediate the associations

between ancestry and outcomes or that ancestry is a non-causal correlate of the trait in question that's actually caused by something else if the mediation is not spurious. Ultimately, controls alone cannot establish causality [50].

Four studies we know of have related genetically-assessed ancestry to cognitive ability in samples of African-Americans [24,25,51,52]. Others have done this for Hispanic Americans [24,25] and various groups in Latin America [53–55]. Of these, the earlier ones [51–54] relied on ancestry estimates derived from blood groups. The use of blood groups has been criticized on the grounds that they are unable to reliably assess global ancestry [11,27].

A recent study by Kirkegaard et al. [24] focused on the relationship between genetic ancestry and *g*. The authors analyzed the multisite Pediatric Imaging, Neurocognition, and Genetics (PING) dataset. They found that European ancestry was associated with *g* among both self-identified African and Hispanic Americans (respectively, $r = 0.20$, $N = 225$; $r = 0.23$, $N = 328$; Tables S10–S11). They assessed genetic ancestry with 15,000 SNPs, making their index highly-reliable [56]. Ancestry explained both the full gap between African and European-Americans and most of the gap between Hispanic and European-Americans in *g*. However, the analysis had a few shortcomings. First, it was based on relatively small samples. Second, the heritability of *g* was unknown. Since the sample comprised children (with a mean age of 12 years) this is potentially concerning due to the Wilson effect. Third, strict factorial invariance (SFI) was not established so it cannot be unequivocally said that the group differences in *g* were measurement invariant. (In our supplementary materials we show that SFI along with the strong version of Spearman's hypothesis hold in the PING data.) Fourth, the sample was geographically dispersed. Such geographic dispersion could lead to spurious correlations between ancestry and cognitive ability [57], though the location of the MRIs did not affect estimates. Finally, as the authors note, no measure of skin color was included in the study, opening up the results to confounding by phenotype-based discrimination.

These results have been replicated in a preregistered study relating European ancestry to cognitive ability in a sample of African and Hispanic Americans ($r = 0.297$, $N = 193$, [25]). This sample was composed of adults, and used 27 items from the International Cognitive Ability Resource Team [58] as a cognitive ability measure. This analysis also had shortcomings. For one, it was on a relatively small sample of individuals recruited online through Qualtrics (an online pollster) and participants self-reported their ancestry (as assessed by commercial DNA testing services such as Ancestry.com or 23andMe), opening these up to reporting error and bias due to differences in admixture estimates from different companies. Additionally, SES was not measured. Moreover, as with Kirkegaard et al. [24], the heritability in this sample is unknown, there was no measure of skin color, and the sample was geographically dispersed.

The purpose of the present study is to conduct an admixture analysis capable of addressing the aforementioned problems with previous studies. We focus on African and European-Americans because the former represent a population substantially admixed with the latter. Generally, African-Americans have around 20% European DNA [37,59–62]. Provided that our African-American sample is sufficiently variable in terms of European admixture, we should be able to detect whether an association exists between cognitive ability and admixture from the two parent populations (i.e., Africans and Europeans) and, if so, to what degree genetic admixture mediates the observed mean difference between the groups. Moreover, if the samples are representative of their respective populations (and statistical power is sufficient), a finding of no association would indicate that an evolutionary-genetic explanation of the gap is unlikely [26,63].

The present work uses a population-representative Philadelphia-based sample, the Philadelphia Neurodevelopmental Cohort (PNC), otherwise known as the Trajectories of Complex Phenotypes study (TCP). Due to the location, the results are directly comparable to those of Scarr et al. [52]. Our analysis has numerous advantages compared to earlier admixture studies. First, participants all came from the same location, so geographic confounding is not an issue. Second, we assessed measurement invariance (MI) for the cognitive test battery using multi-group confirmatory factor

analysis (MGCFA; [64]). Third, the heritabilities of the *g* factor and subtest scores have already been estimated for this sample. Specifically, Mollon et al. [65] reported heritabilities for *g* of 0.61 (standard error (S.E.) = 0.14) and 0.72 (S.E. = 0.07) for the non-Hispanic African and European-Americans in this sample respectively. Fourth, we included estimates of skin, hair, and eye color to evaluate phenotypic discrimination (i.e., colorism) models of the observed differences. Fifth, we validated polygenic scores (PGS) associated with cognitive ability for both the African- and European-American samples and we examined to what extent cognitive ability- and education-related PGS (eduPGS), could account for group differences. Sixth, we tested for Jensen effects in relation to ancestry, heritability, and eduPGS. Seventh, we examined whether MI was tenable across the full range of European ancestry using local structural equation modeling (LSEM).

2. Materials and Methods

The TCP study was originally intended to evaluate behavioral dimensions predicting risk of mental illness [66,67]. The total sample includes data from 9421 genotyped participants assessed primarily from 2010 to 2013. Demographically, the sample was 51.7% female, 55.8% European-American, 32.9% African-American, and 11.4% Other, with a mean age of 14.2 (standard deviation (SD) = 3.7) years of age. Participants were recruited from the Philadelphia area. Persons with severe cognitive or medical impairments were excluded from the final sample. The subjects were English-speaking people aged 8–21 years at the time of testing. Access to the data was obtained through dbgap (phs000607.v3.p2) via Bryan J. Pesta.

2.1. Cognitive Ability

Participants were administered the Penn Computerized Neurocognitive Battery (PCNB; [68]). This battery was built to be highly-reliable, psychometrically-robust, and to incorporate tasks linked to specific brain systems. The PCNB consists of 14 tests grouped into five broad behavioral domains: Executive Control, Episodic Memory, Complex Cognition, Social Cognition, and Sensorimotor Speed. The tests in the battery are as follows: Penn Conditional Exclusion Test (PCET; meant to assess Mental Flexibility), Penn Continuous Performance Test (PCPT; Attention), Letter N-Back Task (LNB; Working Memory), Penn Word Memory Task (PWMT; Verbal Memory), Penn Face Memory Task (PFMT; Face Memory), Visual Object Learning Test (VOLT; Spatial Memory), Penn Verbal Reasoning Test (PVRT; Language Reasoning), Penn Matrix Reasoning Test (PMRT; Nonverbal Reasoning), Penn Line Orientation Test (PLOT; Spatial Ability), Penn Emotion Identification Test (PEIT; Emotion Identification), Penn Emotion Differentiation Test (PEDT; Emotion Differentiation), Motor Praxis Test (MP; Sensorimotor Speed), Finger Tapping (Tap; Sensorimotor Speed), and the Penn Age Differentiation Test (PADT; Age Differentiation). The sample also completed the Wide Range Achievement Test (WRAT), which is a highly-reliable broad ability measure [67].

Approximately 1.4% of the cognitive data were missing. We excluded individuals who did not have data for at least half the tests ($n = 115$). We imputed values for the remaining cases using IRMI (iterative robust model-based imputation) [69,70]. This left us with complete cognitive data for 3,067 African- and 5,076 European-Americans. Since we are only concerned with African and European-Americans in this study we report results only for these groups. We residualized cognitive test variables for age and sex using a natural spline model.

In order to ensure this battery was unbiased, we followed the theoretical configuration reported by Moore et al. (2016) [68] and performed an MGCFA [64,71,72] with our two groups being self-identified African- and European-Americans. The group factors we used were Executive Functioning (EF), Episodic Memory (EM), and Complex Reasoning (CR). The tests which loaded onto these factors were the LNB, PCET, PCPT (EF), PFMT, PWMT, VOLT (EM), WRAT, PVRT, PLOT, and PMAT (CR). We attempted, alternatively a five factor model but the Social Cognition (PEIT, PEDT, PADT) and Sensorimotor Speed (MP, Tap) factors had low loadings and identification issues so we removed these tests.

The three-factor baseline model was found to fit well in both groups and the addition of a bifactor g considerably improved model fit. SFI was found for this model. The strong and contra forms of Spearman's hypothesis [2] were rejected but the weak model fit the data well. Our constraint for testing the weak model was to set the episodic memory factor mean to 0 while the other group factor means were freed. The resulting mean differences appear in Table 1.

Table 1. Factor score differences between African and European-Americans based on the Weak Spearman's Hypothesis Model.

| Factor | Estimate | SE | Lower 95% CI | Upper 95% CI |
|-----------------------|----------|-------|--------------|--------------|
| g | 1.046 | 0.020 | 1.007 | 1.085 |
| Complex Reasoning | 0.356 | 0.026 | 0.305 | 0.407 |
| Executive Functioning | −0.055 | 0.028 | −0.109 | −0.0001 |
| Episodic Memory | 0 | – | – | – |

Note: Positive values indicate higher European-American scores and vice-versa. SE = standard error.

These results accord with previous analyses (e.g., [2,73]) in that there is a nearly one d difference in cognitive ability between African and European-Americans and that this difference is concentrated on the g factor. The model without constraints for Spearman's hypothesis shows that (net of the g factor) there is an African-American advantage in executive functioning and episodic memory. This replicates patterns found in prior studies (e.g., [18,73]). The MGCFA results are discussed more fully in the Supplementary Materials. For the admixture analysis, we used g scores since differences were primarily relegated to this factor.

2.2. Parental Socioeconomic Status

Only paternal and maternal education were available to us as measures of SES. We coded parental education as the average of each, both z-scored first. When data were only available from one parent this value was used. The final score was then z-scored again. This variable was used as a control for SES in our admixture regressions but because we were unable to factor analyze a variety of SES measures and extract a more reliable indicator [74] the control can only be regarded as partial. With this noted, parental education has been found to be more important than wealth in shaping children's intelligence [75]. The effect of SES as a background variable in our MGCFA is investigated in our Supplementary Materials.

2.3. Self-Identified Race/Ethnicity (SIRE)

Subjects chose their self-identified race/ethnicity (SIRE) based on yes/no questions wherein it was possible to select multiple races or ethnicities. The choices were: Black or African-American; American Indian or Alaskan Native; Asian; European-American; Hispanic/Latino; Native Hawaiian/Pacific Islander; Other; and Not Available/Pending Validation. We coded European-American following standard procedures: subjects who selected the European-American option only and did not report being Hispanic. African-American was coded the same way. Finally, we coded subjects who selected both European- and African-American as biracial African-European-American (AA|EA). We then filtered subjects with different identifications, reducing the sample size from an initial N of 9496 to 8580. Of the included participants, there were 5183 European-Americans, 3155 African-Americans, and 242 biracial African-European-Americans.

2.4. Genetic Ancestry Percentages

Satterthwaite et al. [76] described the genotyping of the PNC dataset. Briefly, the participants were genotyped on six different arrays from two different manufacturers. The different arrays covered different variants. As such, it was necessary to impute them to obtain large, overlapping sets of single nucleotide polymorphisms (SNPs). We imputed the dataset using the Michigan Imputation Server

(<https://imputationserver.sph.umich.edu/index.html>) with the Minimac3 imputation algorithm, 1000G Phase 3 v5 as reference panel, and Eagle v2.3 Phasing. In total, 15.5 M variants were available for analysis. To reduce computation time, we filtered these to variants with a minor allele frequency (MAF) of at least 0.05 in the subset of interest (i.e., the combined sample of European and African-Americans). That is, we selected only variants for which the second-most common alleles occurred in the population at a frequency of 0.05 or higher. This screening left 6.5M variants to compute genetic ancestry percentages. Note, this was only done in the context of computing admixture scores. We did not filter for MAF when computing color scores, as we did not want to lose variants that affected them. These operations were done using PLINK v1.90b6.8 [77]. To estimate individual global ancestry we used ADMIXTURE version 1.3.0 D.H. [78]. Since we were only concerned with European and African-Americans, we ran ADMIXTURE with $k = 2$ genetic clusters. Some subjects either had no genotypes available or their data failed quality control. Thus, the final sample size was reduced to 7399. Consistent with previous research (e.g., [59], SIRE is strongly associated with genetic ancestry. This can be seen in Figure 1 which shows the probability of identifying with a particular SIRE group as a function of European admixture.

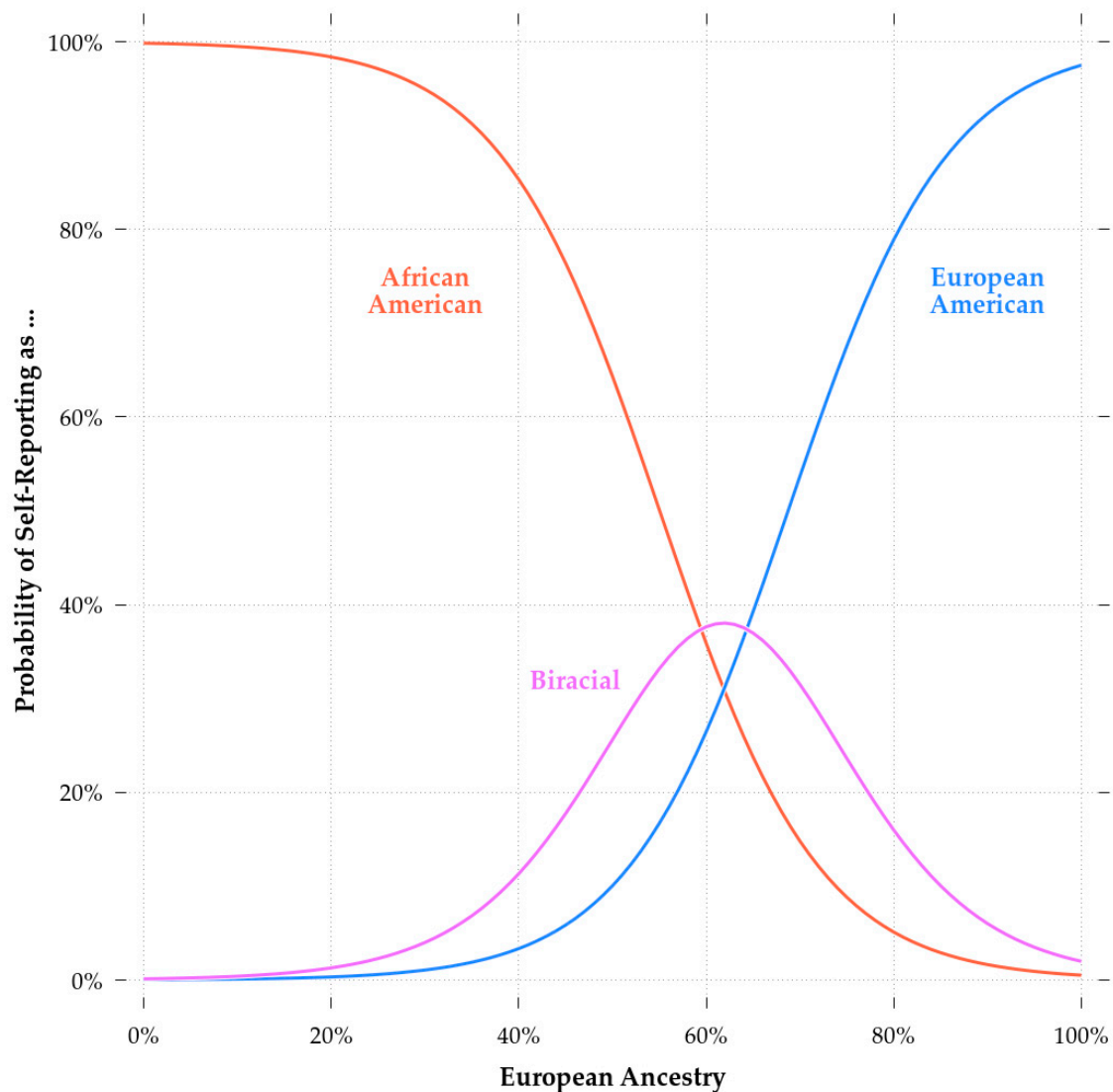


Figure 1. Probability distribution for identifying as African-, biracial African-European, and European-American as a function of genetic ancestry for the Trajectories of Complex Phenotypes (TCP) sample.

2.5. Skin, Hair, and Eye Color

The data did not include measures of appearance, so we opted to impute these based on genotypes. We used the HirisPlex-S web application to do this (<https://hirisplex.erasmusmc.nl/>). This application was developed by the U.S. Department of Justice for use in forensic investigations. It imputes skin, hair, and eye color probabilities with a high degree of accuracy based on 41 SNPs (with overlapping variants; 6 for eye color, 22 for hair color, and 36 for skin color). This tool has been validated on thousands of people from diverse regions of the world [79].

We focus on skin color since this trait is given primacy by colorist theorists (e.g., [80,81]) and because we were able to calculate skin color scores for a larger subsample than for hair or eye color. HirisPlex-S gives probabilities of the Fitzpatrick Scale skin type (i.e., Type I, scores 0–6, “palest, freckles”; Type II, scores 7–13; Type III–IV (combined), scores 14–27; Type V, scores 28–34; Type VI, scores 35–36, “deeply pigmented dark brown to darkest brown”). We weighted the medium score of each Type (e.g., Type I = 3) by the probability of each type in order to create a single color measure. Unfortunately, because of the poor tagging of the relevant variants in some of the arrays, the imputed phenotypes were only available for a subset of the data ($N = 7321$ with cognitive ability and ancestry, versus $N = 5533$ with cognitive ability, ancestry, and skin color). By this scale, higher scores indicate darker colors.

The correlation between our imputed color score and European ancestry was -0.87 for the combined sample ($N = 5585$) and -0.39 for the African-Americans only sample ($N = 1557$). Figure 2 shows the regression plot for skin color and European ancestry, with SIRE groups marked in red (African-American), green (biracial African-European-American), and blue (European-American). The correlation found among African-Americans is similar to those previously reported (r 's 0.25 to 0.71; median: ~ 0.50) for African-Americans and other groups of African descent [56,61,82,83]. Furthermore, the predicted skin color values matched the expected values on the Fitzpatrick scale. The mean score for European-Americans was 14.70, placing them in category III (i.e., sometimes mild burn, tans uniformly) bordering on category II (i.e., usually burns, tans minimally), and for African-Americans, 30.96, placing them in category V (very rarely burns, tans very easily).

2.6. Cognitive Ability-Related Polygenic Scores

We scored the genomes using PLINK v1.90b6.8 [77] based on the genome-wide association study (GWAS) results for educational attainment reported in Lee et al. [84]. We constructed four different PGS based on the relative trade-off between differential linkage disequilibrium (LD) decay bias and the within-population signal. For background, a PGS “represents an aggregation, usually in the form of a sum, of the estimated effect sizes of the genetic variants in the genome” [85]. LD is the non-random association of alleles at different loci in a particular population. LD decay is the breakdown of this association between divergent populations. LD decay bias is bias in PGS—which are calculated based on both causal variants and variants in linkage with causal and non-causal ones—due to populations having different patterns of associations between variants and sites that are nearby (or in linkage).

First, we used the eduPGS with all variants trained without the 23andMe cohort ($n = 7,762,369$ SNPs overlapping in the present dataset) to construct a predictor with good within-population validity but very high LD decay bias [86,87]. Second, we used the multi-trait analysis of genome-wide association study (MTAG) eduPGS 10k SNPs ($n = 8442$ overlapping variants) to construct a predictor with good within-population validity but high LD decay bias. The MTAG eduPGS were PGS computed using MTAG. This is a method for analyzing statistics from genome-wide association study (GWAS) on different but genetically correlated traits (e.g., education and intelligence). Third, we used the MTAG eduPGS lead SNPs to construct an eduPGS with moderate within-population validity and LD decay bias ($n = 1558$ overlapping variants). The lead SNPs are the ones which had the most-significant association with education/intelligence within each cluster of LD-associated SNPs. The reasoning is that because SNPs with lower p -values (i.e., greater statistical significance) are more likely to be causal [88], and because causal SNPs are more likely to be transethnically valid [86,89], using SNPs

with a higher significance may increase the transethnic validity of eduPGS [90]. Fourth, we used Lee et al.'s [84] putatively causal variants to construct an eduPGS with minimal LD decay bias and low within-population validity ($n = 111$ overlapping variants).

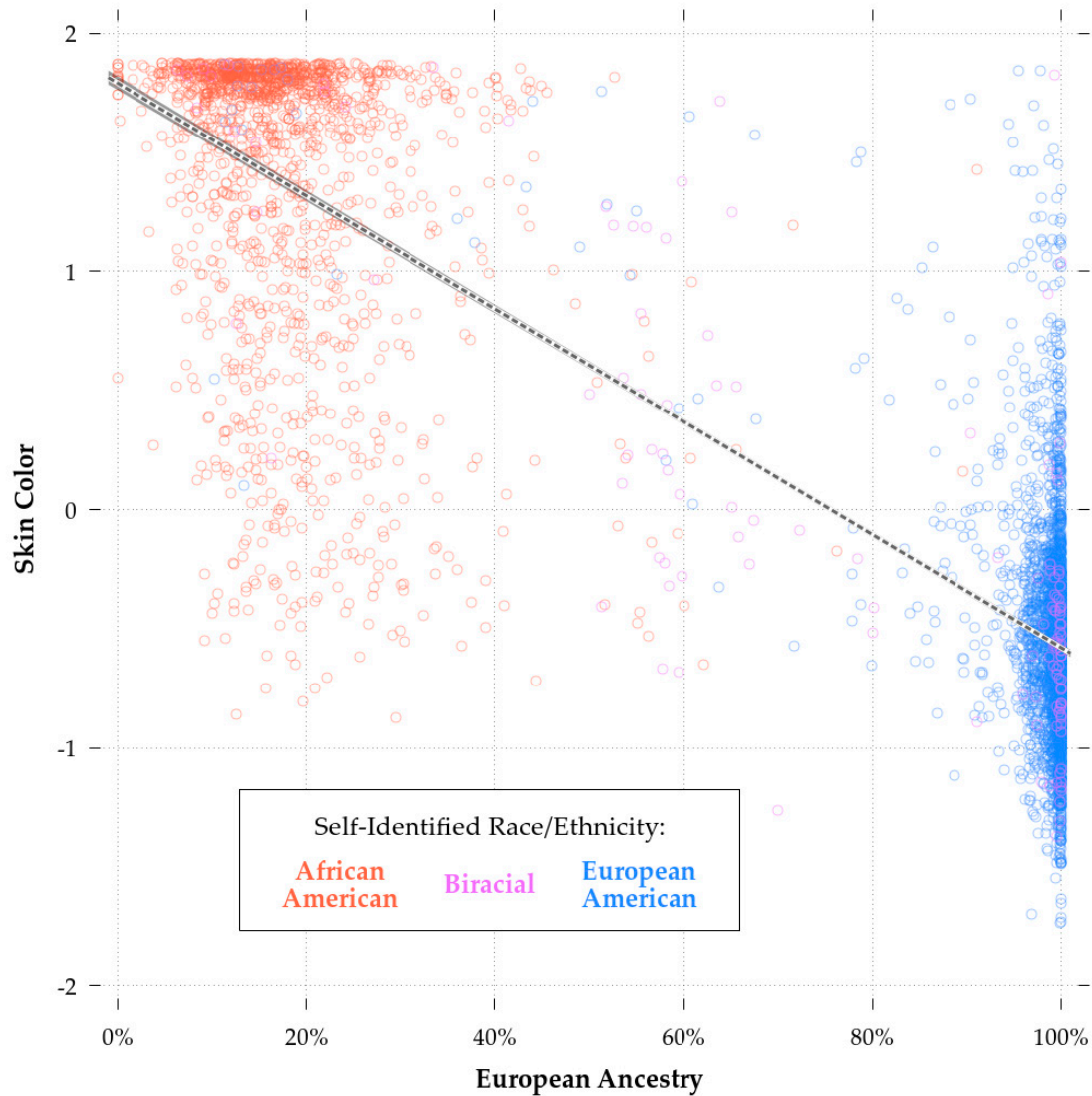


Figure 2. Regression plot of the relation between color (with higher values indicating darker color) and European genetic ancestry.

3. Results

3.1. Descriptive Statistics

Table 2 shows descriptive statistics for all groups. AA/EAs were primarily European (80%) in ancestry. Correspondingly, they were closer to the EA means on SES, g , and color. The AA/EA g difference was $d = 0.984$ ($IQ_{EA} = 100$ versus $IQ_{AA} = 85.28$, with the biracial group scoring in-between at 97.96), while the SES difference was $d = 0.99$ and the difference in color was $d = 3.60$.

Table 2. Sample characteristics for the African, European, and biracial African-European-American participants.

| | Age (SD; N) | % European (SD; N) | Mean SES (SD; N) | Mean <i>g</i> Score (SD; N) | Mean Color (SD; N) |
|------------------|--------------------|---------------------|--------------------|-----------------------------|--------------------|
| African | 14.08 (3.75; 2227) | 0.187 (0.117; 2228) | −0.57 (0.77; 2180) | −1.01 (1.07; 2179) | 30.96 (5.87; 1557) |
| Biracial | 13.15 (3.58; 232) | 0.796 (0.289; 232) | 0.16 (0.95; 230) | −0.14 (1.05; 228) | 19.18 (7.61; 166) |
| African-European | 13.76 (3.64; 4937) | 0.986 (0.059; 4939) | 0.33 (0.96; 4909) | 0.00 (1.01; 4914) | 14.70 (3.84; 3862) |

Note: Standard deviations and sample sizes appear in parentheses. SES = socioeconomic status.

3.2. Bivariate Relationships among the Variables

Correlations between *g*, parental SES, genetic ancestry, and SIRE group appear in Tables 3 and 4. Correlations are as expected based on the cognitive and SES differences between different SIRE groups and the strong correlation between genetic ancestry and SIRE. However, this bivariate analysis is not very informative as outcomes are confounded with non-genetic causes and ancestry variables are negatively correlated by design since individual ancestry must sum to one. The relationship between parental SES and cognitive ability is likely stronger here than is typically reported (see [91]; $r = 0.29$) because we used both parents' education and a latent cognitive ability measure which reduces error that would normally attenuate the strength of this relationship.

Table 3. Pairwise correlations among African-, African-European, and European-Americans.

| | Cognitive Ability | SES | Euro. Ancestry | Afr. Ancestry | SIRE EA | SIRE AA | Color | EduPGS |
|-------------------|-------------------|---------------|----------------|---------------|---------------|---------------|---------------|--------|
| Cognitive Ability | 1 | | | | | | | |
| SES | 0.406 (7253) | 1 | | | | | | |
| European Ancestry | 0.411 (7321) | 0.412 (7319) | 1 | | | | | |
| African Ancestry | −0.411 (7321) | −0.412 (7319) | −1.000 (7399) | 1 | | | | |
| SIRE EA | 0.408 (7321) | 0.413 (7319) | 0.964 (7399) | −0.964 (7399) | 1 | | | |
| SIRE AA | −0.387 (7321) | −0.395 (7319) | −0.928 (7399) | 0.928 (7399) | −0.930 (7399) | 1 | | |
| Color | −0.359 (5534) | −0.354 (5530) | −0.875 (5585) | 0.875 (5585) | −0.838 (5585) | 0.812 (5585) | 1 | |
| EduPGS | 0.402 (7321) | 0.445 (7319) | 0.672 (7399) | −0.672 (7399) | 0.645 (7399) | −0.630 (7399) | −0.614 (7399) | 1 |

Note: All values significant at $p < 0.0001$. Pairwise *N* in parentheses. SES = socioeconomic status, SIRE = self-identified race/ethnicity, eduPGS = education polygenic score.

Table 4. Pairwise correlations among self-identified monoracial African-American (below the diagonal) and self-identified monoracial and biracial African-European-American (above the diagonal).

| | Cognitive Ability | SES | Euro. Ancestry | Afro. Ancestry | Color | EduPGS |
|-------------------|-------------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Cognitive Ability | 1 | 0.315 * (2367) | 0.251 * (2407) | −0.251 * (2407) | −0.188 * (1690) | 0.212 * (2407) |
| SES | 0.256 * (2140) | 1 | 0.276 * (2410) | −0.276 * (2410) | −0.184 * (1690) | 0.244 * (2410) |
| European Ancestry | 0.086 * (2179) | 0.054 * (2180) | 1 | −1.000 * (2460) | −0.639 * (1723) | 0.522 * (2460) |
| African Ancestry | −0.086 * (2179) | −0.054 * (2180) | −1.000 * (1854) | 1 | 0.639 * (1723) | −0.522 * (2460) |
| Color | −0.084 * (1526) | −0.047 (1524) | −0.389 * (1557) | 0.389 * (1557) | 1 | −0.394 * (1723) |
| EduPGS | 0.112 * (2179) | 0.119 * (2180) | 0.328 * (2228) | −0.328 * (2228) | −0.187 * (1557) | 1 |

Note: * Significant at $p < 0.01$. Pairwise *N* in parentheses. SES = socioeconomic status, eduPGS = education polygenic score.

The correlations between cognitive ability and both color and African ancestry are highest in the combined African-, African-European-, and European-American sample, intermediate in the combined mono- and bi-racial African-American sample, and lowest in the monoracial African-American sample. The difference in the magnitudes of the correlations is a result of the first sample having the most variability in ancestry and the last sample having the least. This difference is mostly a statistical artifact of restriction of range, which attenuates correlations [92].

3.3. Regression Analyses

Our primary focus is whether genetic ancestry predicts cognitive ability. The rationale for the analysis has been explained in detail by Kirkegaard et al. [24]. In order to determine if this is the case, we constructed a series of regression models. These models were applied to the African-American and the combined African-, European-, and biracial African-European-American sample. Restricting the first analysis to SIRE African-Americans approximates the methodology used by Scarr et al. [52]. Our initial analysis used European ancestry (EUR) as the independent variable. We then added skin color to test discrimination (i.e., colorism) and other appearance-based hypotheses. Finally, we added SES to assess the effect of familial environmental influences on the observed gap, noting again that these measures are genetically confounded.

To provide results that can be easily compared across groups and studies, we standardize all values except European ancestry and then report the unstandardized beta coefficients. Doing so leaves only ancestry unstandardized. This allows one to interpret the beta coefficient for ancestry as a change in a standard deviation of cognitive ability over a change of 100% European ancestry. Following Kirkegaard et al. [24], we do not report standardized betas (except in Table 5 for illustration), because, in the context of the effect of ancestry on outcomes, these are difficult to compare across groups. This difficulty arises because groups differ substantially in their variability in ancestry and its effects. As such, a change of one standard deviation of ancestry from one group has a substantially different meaning than the same from another group.

In models that only included monoracial African-Americans, we found that European ancestry was always strongly and significantly related to cognitive ability. Skin color (assessed genetically with the highly accurate predictor [79,93]) was associated with cognitive ability (Model 1b, Table 5), but made no significant incremental contribution when ancestry was also in the model (Model 2, Table 5). Results could still be due to phenotypic confounding from other appearance variables. To test this possibility, we fitted a number of models including skin, hair, and eye color. We found that none of these features had significant effects on their own, except for brown eye color, which was positively related to cognitive ability, but with a large standard error. These results are shown in the R notebook. The last monoracial African-American model (Model 3, Table 5) included SES, which had a considerable effect on cognitive ability. However, the so-called sociologist's fallacy [2,5] may be at play here. That is, controlling for parental SES also controls for genetic effects on SES which may be shared with cognitive ability.

As an alternative to the model with only monoracial African-Americans, we next included both individuals who identified as monoracial African- and biracial African-European-American. This is equivalent to the "broadly-defined" African-American group used by Kirkegaard et al. [24]. These results are shown in Table 6. In Model 2, with self-identified European-American race (which, in this case, indicates biracial status) as a covariate, the beta for European ancestry ($B_{EUR} = 0.85$; $n = 2407$) was comparable although slightly higher than that for the monoracial African-American sample ($B_{EUR} = 0.78$; $n = 2179$).

Table 5. Regression analysis for European ancestry as a predictor of g among monoracial African-Americans with controls for skin color (Model 2), and SES (Model 3) added.

| | Model 1 | | | Model 1b | | | Model 2 | | | Model 3 | | |
|-------------------------|----------|------|----------|-----------|------|-----------|----------|------|----------|----------|------|----------|
| Predictor | <i>B</i> | SE | β | <i>B</i> | SE | β | <i>B</i> | SE | β | <i>B</i> | SE | β |
| Intercept | −1.16 | 0.04 | −1.01 | −0.82 | 0.06 | −0.997 | −1.05 | 0.09 | −0.996 | −0.83 | 0.09 | −0.995 |
| EUR | 0.78 *** | 0.19 | 0.09 *** | | | | 0.83 *** | 0.24 | 0.10 *** | 0.67 ** | 0.23 | 0.08 ** |
| Skin Color | | | | −0.13 *** | 0.04 | −0.09 *** | −0.07 | 0.04 | −0.05 | −0.06 | 0.04 | −0.04 |
| SES | | | | | | | | | | 0.36 *** | 0.03 | 0.28 *** |
| Adjusted R ² | 0.007 | | | 0.006 | | | 0.013 | | | 0.082 | | |
| N | 2179 | | | 1526 | | | 1526 | | | 1500 | | |

Note: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Model 1b shows the results with color as an alternative predictor. EUR = European ancestry. SES = socioeconomic status.

Table 6. Regression analysis for European ancestry as a predictor of g among monoracial and biracial African-European-Americans with controls for SIRE (Model 2), skin color (Model 3b), and SES (Model 4) added.

| | Model 1 | | Model 2 | | Model 3a | | Model 3b | | Model 4 | |
|-------------------------|----------|------|----------|------|-----------|------|----------|------|---------|------|
| Predictor | B | SE | B | SE | B | SE | B | SE | B | SE |
| Intercept | −1.22 | 0.03 | −1.17 | 0.04 | −0.62 | 0.05 | −1.07 | 0.08 | −0.83 | 0.08 |
| EUR | 1.21 *** | 0.09 | 0.85 *** | 0.15 | | | 0.80 *** | 0.19 | 0.55 ** | 0.19 |
| SIRE: EA | | | 0.35 ** | 0.12 | | | 0.27 * | 0.13 | 0.18 | 0.13 |
| Skin Color | | | | | −0.25 *** | 0.03 | −0.06 | 0.04 | −0.05 | 0.04 |
| SES | | | | | | | | | 0.37 | 0.03 |
| Adjusted R ² | 0.063 | | 0.066 | | 0.035 | | 0.065 | | 0.134 | |
| N | 2407 | | 2407 | | 1690 | | 1690 | | 1664 | |

Note: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. EUR = European ancestry. SES = socioeconomic status, SIRE = self-identified race/ethnicity.

Comparing Model 2 and Model 3b, biracial status had a significant effect independent of European ancestry. This could be due to selective patterns of self-identification, whereby choosing to report that one is a European-American, regardless of one's genetic ancestry, is associated with greater cognitive ability. This appears to have been the case as evidenced by Figure 3, which shows the relationship between European ancestry and cognitive ability. Being above the regression line was associated with a greater likelihood of biracial African-European self-identification regardless of one's actual ancestry. Whatever the case, unlike with self-reported European-American race, skin color did not have significant predictive validity independent of European ancestry. The comparison between Model 3a and Model 3b shows that the inclusion of ancestry reduced the effect of color from highly significant to non-significant ($B_{\text{color}} = -0.25$ in 3a and $B_{\text{color}} = -0.06$ in 3b, $n = 1,690$). As in the models for monoracial AAs, the inclusion of parental SES reduced, but did not eliminate, the effect of European ancestry (Model 4).

The next set of models utilized the whole sample of African, European, and biracial African-European-Americans. These results appear in Table 7. The first model revealed a substantial impact of European ancestry on cognitive ability. The next model found the same, but also produced a significant effect of SIRE European-American (but not SIRE African-American) self-identification. That is, some incremental validity existed which was associated with self-identification as European-American and this did not occur for self-identification as African-American. The next model included physical appearance in the form of skin color. As with the African-American-only and the combined African- and biracial African-European-American sample, this variable had no significant effect on cognitive ability in the full sample with ancestry included (Model 3b). Our final model included SES. The inclusion of SES reduced the effect of SIRE European-American, although it still remained significant. Nonetheless, in this model, European ancestry was the dominant determinant of cognitive ability. The effect of SES on cognitive ability was essentially the same in the full sample as it was in the monoracial African-American model. The fact that the effect of SIRE European-American weakened with the inclusion of SES is perhaps notable, as it may imply that selective self-identification as European-American is driven in part by social processes. It is clear here though that self-identification and other-identifiable physical characteristics are much less important as determinants of cognitive ability than is genetic ancestry.

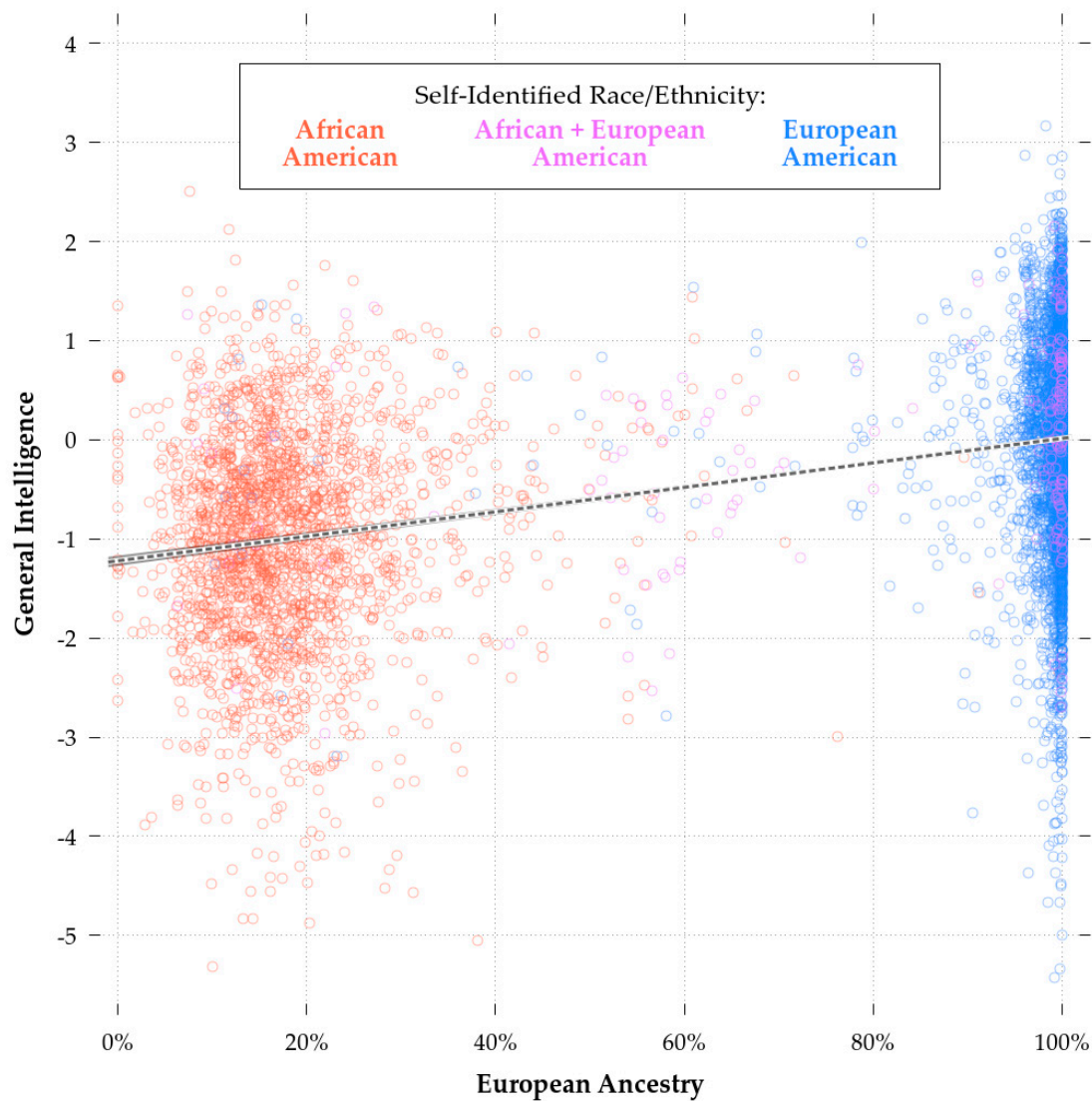


Figure 3. Regression plot for the relation between g and European ancestry ($r = 0.411$).

Table 7. Regression analysis for European ancestry as a predictor of g among African-, European-, and biracial African-European-Americans with controls for SIRE (Model 2) skin color (Model 3), and SES (Model 4) Added.

| | Model 1 | | Model 2 | | Model 3a | | Model 3b | | Model 4 | |
|-------------------------|----------|------|----------|------|-----------|------|----------|------|----------|------|
| Predictor | <i>B</i> | SE | <i>B</i> | SE | <i>B</i> | SE | <i>B</i> | SE | <i>B</i> | SE |
| Intercept | -1.22 | 0.03 | -1.15 | 0.09 | -0.28 | 0.01 | -1.09 | 0.11 | -0.91 | 0.11 |
| EUR | 1.23 *** | 0.03 | 0.75 *** | 0.13 | | | 0.74 *** | 0.16 | 0.59 *** | 0.15 |
| SIRE: EA | | | 0.41 *** | 0.11 | | | 0.35 ** | 0.12 | 0.22 * | 0.11 |
| SIRE: AA | | | 0.00 | 0.08 | | | -0.02 | 0.09 | 0.01 | 0.08 |
| Skin Color | | | | | -0.40 *** | 0.01 | -0.02 | 0.03 | -0.01 | 0.03 |
| SES | | | | | | | | | 0.34 *** | 0.01 |
| Adjusted R ² | 0.169 | | 0.170 | | 0.129 | | 0.166 | | 0.238 | |
| N | 7321 | | 7321 | | 5534 | | 5534 | | 5488 | |

Note: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. EUR = European ancestry. SES = socioeconomic status, SIRE = self-identified race/ethnicity.

Cumulatively, European ancestry was consistently positively related to cognitive ability in all models. SES was similarly related to cognitive ability. European-American SIRE also had a significant

effect independently of genetic ancestry, indicating that differences were not wholly explained by ancestry. Skin color was not significantly related to cognitive ability in any of our models which included genetic ancestry. However, it was related to cognitive ability in models not including ancestry, as found in previous studies [17,94–96] (for a review of older studies, see [1]).

Recent immigrants from Africa in the sample may have affected the scores reported here as migrants may have been selected for cognitive ability when they emigrated from their home countries. As such, we conducted a robustness test by rerunning the analyses excluding individuals with one percent or less European ancestry. This exclusion did not substantially affect the results (reported in the R notebook, Model 17b). Thus, confounding due to migration likely did not influence our findings.

3.4. Cognitive Ability and Education-Related Polygenic Scores (eduPGS)

We examined the validity of four cognitive ability and education-related PGSs reported by Lee et al. [84]. The eduPGS were derived from European populations. First, we used the eduPGS based on all variants trained without the 23andme cohort ($n = 7,762,369$ variants overlapping in the present dataset). The goal was to construct a predictor with good within-discovery population (European, in this case) validity, but very high LD decay bias, and thus low transethnic validity. Second, we used MTAG eduPGS with 10k variants ($n = 8442$ overlapping variants) to construct a predictor with good within-discovery population validity and reduced LD decay bias. Third, we used the MTAG eduPGS lead SNPs ($n = 1558$ overlapping variants) to see if we could reduce LD decay bias and increase transethnic validity while retaining within-discovery population validity. Finally, we used the putatively causal variants ($N = 111$ overlapping variants in our dataset) to construct an eduPGS with minimal LD decay bias, but low within-discovery population validity. At some point, screening SNPs based on p values may lead to lower validity in both the discovery population and other-ethnic populations, despite having relatively high transethnic validity [90]. The goal is to identify PGS which have both high within-discovery population and transethnic validities.

The correlations with g are shown in Table 8, with results for European-Americans above the diagonal and results for (monoracial) African-Americans below. Since the PNC is independent from the samples analyzed by Lee et al. [84], the results for European-Americans constitute an independent replication of those reported by Lee et al. [84]. This may be of interest to some because our dependent variable was a good measure of g .

Table 8. Pairwise correlations between cognitive ability and education/intelligence-related polygenic scores (European-American above the diagonal, African-American below).

| | Cognitive Ability | Putative Causal | GWAS_edu PGS | MTAG_10K_eduPGS | MTAG_Lead eduPGS |
|-------------------------|-------------------|------------------|------------------|------------------|------------------|
| Cognitive Ability | 1 | 0.058 *** (4914) | 0.225 *** (4914) | 0.227 *** (4914) | 0.210 *** (4914) |
| Putative Causal_edu PGS | 0.031 (2179) | 1 | 0.216 *** (4939) | 0.315 *** (4939) | 0.348 *** (4939) |
| GWAS_edu PGS | 0.044 * (2179) | 0.135 *** (2228) | 1 | 0.645 *** (4939) | 0.574 *** (4939) |
| MTAG_10K_eduPGS | 0.112 *** (2179) | 0.228 *** (2228) | 0.484 *** (2228) | 1 | 0.837 *** (4939) |
| MTAG_Lead_PGS | 0.094 *** (2179) | 0.266 *** (2228) | 0.453 *** (2228) | 0.801 *** (2228) | 1 |

Note: * Significant at $p < 0.05$; ** Significant at $p < 0.01$; *** Significant at $p < 0.001$. N in parentheses. GWAS = genome-wide association study (standard one-at-a-time regression), MTAG = multi-trait analysis of GWAS, eduPGS = education polygenic score, lead = genome wide-significant hits only.

Of the PGS, the MTAG_10k PGS had the highest validity in both the European-American and African-American samples, although the validity for African-Americans was reduced by 51%. The results for MTAG_10k and MTAG eduPGS lead SNPs correlated highly with one another ($r > 0.80$) in both the African- and European-American samples. These eduPGS also had similar relationships with cognitive ability.

As expected, the GWAS eduPGS had high within-source population validity but low transethnic validity. While significant in the African-American sample, the validity was only $0.044/0.225 = 20\%$ of

that in the European-American sample. This low transethnic validity occurred because the 7,762,369 variants in conjunction with high LD decay produced considerable noise. Due to being based on only 111 overlapping variants, the putatively causal eduPGS had low validity within both samples but had the highest relative transethnic validity: $0.031/0.058 = 54\%$.

Consistent with theory [90], the eduPGS with the highest validity in both the African- and European-American samples was the MTAG_EA_10K ($r = 0.112$ & $r = 0.227$, respectively). As such, in the following analyses, we restricted consideration to these scores. While retaining some validity in the African-American sample, the MTAG_10k eduPGS showcased predictive bias. Indeed, a Fisher r -to- z Test showed that the differences in the correlations for African and European-Americans was highly significant ($z = -4.6$, $p < 0.01$). This difference is illustrated in Figure 4, which shows the regression plot for eduPGS and g for European (red) and African-Americans (blue).

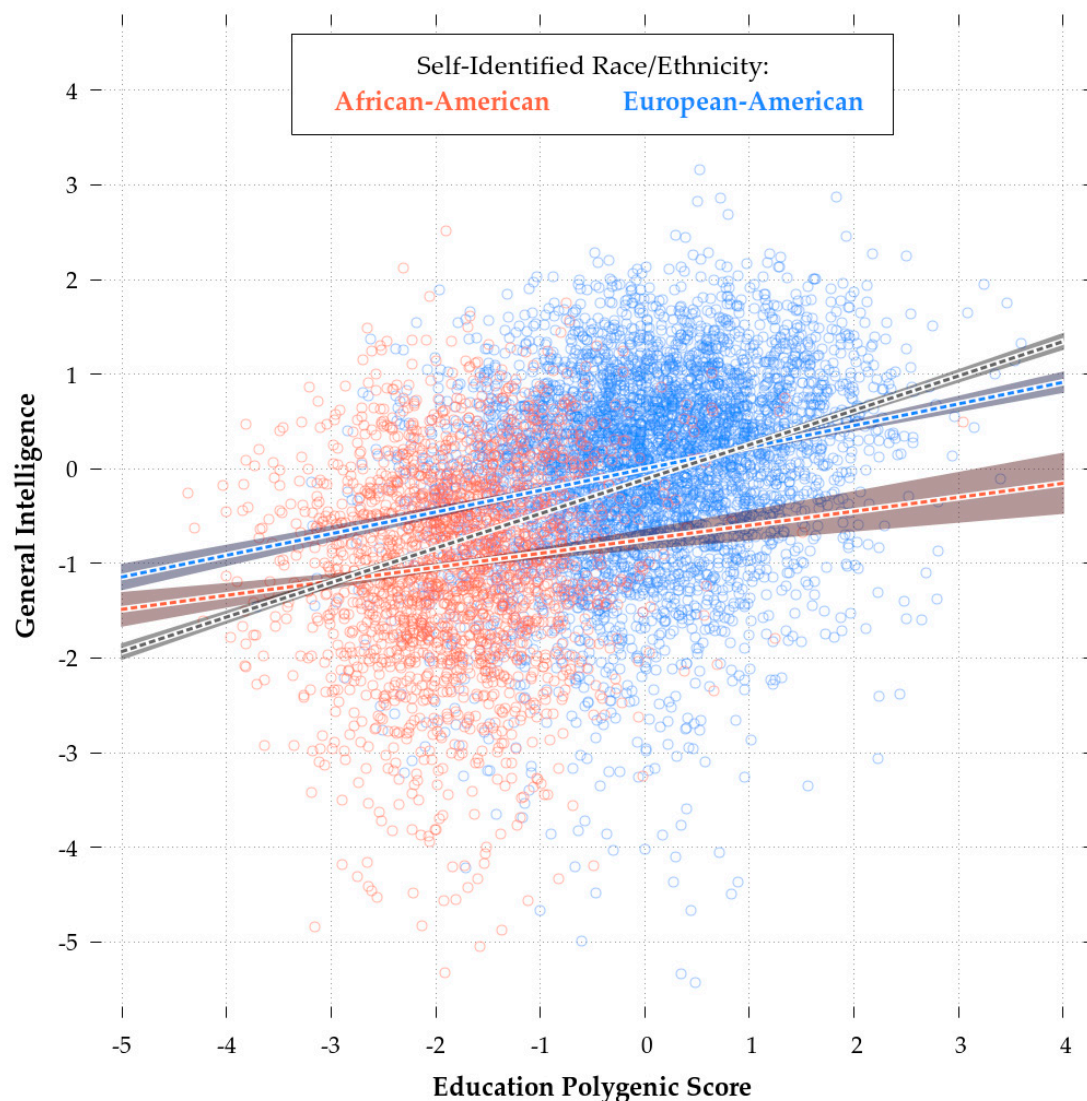


Figure 4. Regression plot for the predictive validity of MTAG 10k eduPGS with Respect to g in the African-American (Red; $r = 0.112$) and European-American (Blue; $r = 0.227$) Samples.

The slope of the regression line and the intercepts for the African-American sample are both lower. As Zanetti & Weale [86] note, differences in SNP predictivity could be due to (1) lack of statistical power, (2) allele frequency differences, (3) linkage disequilibrium, or (4) differences in true causal variant effect sizes/directions. These authors suggest that the last explanation is generally not necessary.

Regardless, a combination of these factors could lead to PGS bias in favor of Europeans (or Africans). There are a number of possible ways in which such bias could have arisen. For example, imagine the case for more recent polymorphisms. These more recent polymorphisms will probably have a higher frequency in the discovery population. If recent selection favored IQ-promoting variants and purged IQ-reducing variants, the more recent polymorphisms will be IQ-promoting, which will result in bias in favor of the discovery population. There is some evidence of an increase in PGS over time [97]. Of course, the reverse would be true, if recent selection disfavored high IQ alleles. However, given that most mutations are deleterious [98], if the PGS contain a large number of recent variants it is likely that they'll be biased against their discovery populations. For disease phenotypes, Kim, Patel, Teng, Berens, & Lachance [99] showed how this bias can function and found that correcting for differences in derived vs. ancestral polymorphisms can improve the transracial predictivity of PGS. In a supplementary analysis, conducted to address a reviewer's point, we did not find evidence of a substantive (or even statistically significant) effect of young vs. old polymorphisms. Thus, the concern raised regarding bias due to polymorphism age (e.g., [99]) does not seem to be a problem in this case and so we did not create polymorphism age-corrected PGS.

Table 9 shows eduPGS means and standard deviations for African, biracial African-European, and European-Americans. The eduPGS difference is large at $d = 1.89$ and it is larger than the phenotypic gap in g . This would be expected even if the PGS were unbiased given the imperfect relationship between PGS and g . Beyond this and possible bias in the PGS, another possibility is that the PGS differences between groups are primarily in common variants, while the differences between individuals within groups are in both common variants and rare variants that arose recently by mutations and which are under mutation-selection balance. Given this scenario, PGS which indexed mostly common variants between populations would manifest as reduced phenotypic differences even assuming consistent validity.

Table 9. Mean racial differences in eduPGS (MTAG_10k).

| | <i>N</i> | <i>M</i> | <i>SD</i> |
|------------------------------------|----------|----------|-----------|
| European-American | 4939 | 0.00 | 1.00 |
| Biracial African-European-American | 232 | −0.59 | 1.15 |
| African-American | 2228 | −1.79 | 0.81 |

The association between eduPGS (MTAG_10k) and g in the African-American sample could, in principle, result from confounding with environmental factors. This could be an indirect effect of the association between g and European ancestry or, alternatively, color. Alternatively, the variants in the eduPGS could be “chopstick genes” [100] which have a statistical but no causal effect. If eduPGS were merely indexing European ancestry, one would expect the GWAS eduPGS (which is based on more than 7 million additional variants) to exhibit an association as high as the one we found with MTAG_EA_10k eduPGS. This is because the GWAS_eduPGS, with its greater number of variants, is likely to be a better index of ancestry. This does not appear to be the case. Regardless, to explore this possibility further, we ran regression models with European ancestry (Model 1b) and color scores (Model 2b) for the monoracial African-American and combined African-, European-, and biracial African-European-American samples. The results are shown in Tables 10 and 11 respectively.

Table 10. Regression results for the effect of eduPGS on cognitive ability among monoracial African-Americans.

| Predictor | Model 1b | | Model 2b | |
|-------------------------|----------|------|----------|------|
| | B | SE | B | SE |
| Intercept | −0.88 | 0.08 | −0.75 | 0.12 |
| EUR | 0.50 * | 0.21 | 0.51 * | 0.25 |
| eduPGS | 0.12 *** | 0.03 | 0.13 *** | 0.04 |
| Color | | | −0.07 | 0.04 |
| Adjusted R ² | 0.014 | | 0.021 | |
| N | 2179 | | 1526 | |

Note: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. EUR = European ancestry. eduPGS = education polygenic score.

Table 11. Regression results for the effect of eduPGS on cognitive ability among African, biracial African-European, and European-Americans.

| Predictor | Model 1b | | Model 2b | |
|-------------------------|----------|------|----------|------|
| | B | SE | B | SE |
| Intercept | −0.73 | 0.09 | −0.64 | 0.12 |
| EUR | 0.29 * | 0.13 | 0.27 | 0.16 |
| SIRE: EA | 0.44 *** | 0.10 | 0.37 ** | 0.11 |
| SIRE: AA | 0.04 | 0.07 | 0.01 | 0.09 |
| eduPGS | 0.21 *** | 0.01 | 0.20 *** | 0.01 |
| Color | | | −0.01 | 0.03 |
| Adjusted R ² | 0.200 | | 0.196 | |
| N | 7321 | | 5534 | |

Note: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. EUR = European ancestry. SES = socioeconomic status, SIRE = self-identified race/ethnicity, eduPGS = education polygenic score.

Adjusting for European ancestry does not reduce the validity of eduPGS in the African-American sample (Table 10; Model 1b), nor does controlling for skin color. In fact, the validity here increases slightly relative to that found among European-Americans. Table 12 shows the regression results for European-Americans (Model 2). Comparing the monoracial African-American SIRE beta (Model 1b, Table 10) to the European-American beta (Model 2, Table 12) gives an African-American validity 52% the size of the European-American validity.

Table 12. Regression results for the effect of eduPGS on cognitive ability among European-Americans.

| Predictor | Model 1 | | Model 2 | | Model 3 | |
|-------------------------|---------|------|----------|------|----------|------|
| | B | SE | B | SE | B | SE |
| Intercept | −0.45 | 0.25 | 0.02 | 0.24 | −0.03 | 0.27 |
| EUR | 0.45 | 0.25 | −0.02 | 0.24 | 0.04 | 0.28 |
| eduPGS | | | 0.23 *** | 0.01 | 0.22 *** | 0.02 |
| Color | | | | | 0.02 | 0.04 |
| Adjusted R ² | 0.000 | | 0.051 | | 0.50 | |
| N | 4914 | | 4914 | | 3844 | |

Note: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. EUR = European ancestry. SES = socioeconomic status, SIRE = self-identified race/ethnicity, eduPGS = education polygenic score.

If we take the beta of 0.124 in the AA-only sample (Model 1b, Table 10) as an estimate of the LD-unbiased effect of eduPGS on g , the g -score differences due to eduPGS would be 0.124×1.89 (i.e., the Cohen's d for the African-/European-American eduPGS difference) = 0.23. This implies that 20%–25% of the difference in g can be accounted for by presently known cognitive ability- and education-related PGS.

Concerns have been raised that the eduPGS computed by Lee et al. [84] may be biased against non-Europeans due to the inclusion of population-specific variants [101] (for a more general discussion see [99]). These population-specific variants would have very low frequency in non-European populations. Following Piffer's suggestion [101], as a robustness check, we computed MTAG eduPGS excluding variants with $MAF < 0.01$ (leaving 7636 overlapping variants) and < 0.05 (leaving 7172 overlapping variants) among African lineages. The 1000 Genomes reference samples were used to determine the African MAF. This exclusion had no substantive effect on the eduPGS validity with respect to g for either African or European-Americans. For SIRE monoracial African-Americans $r_{MAF \geq 0.01} = 0.112$ and $r_{MAF \geq 0.05} = 0.114$.

Theory predicts that, in the absence of countervailing confounds, variants which show concordant as opposed to discordant effects across racial groups, will exhibit higher validity in non-discovery populations (Piffer, personal communication, February 3, 2019; [90]). This is because variants which show transracially concordant effects are more likely to be causal and also because causal variants, as opposed to variants in LD with causal ones, are more likely to have transethnic predictivity [89,102]. These predictions are borne out in this sample. Among European-Americans, the predictivity for concordant MTAG_10k eduPGS was higher ($r_{\text{concordant}} = 0.223$ vs. $r_{\text{discordant}} = 0.145$), and among African-Americans, it was even more so ($r_{\text{concordant}} = 0.223$ vs. $r_{\text{discordant}} = -0.128$). Similarly, when we grouped variants by the p value for the interaction between race and the effect on g (with higher p values signifying less discordance in effect), among African-Americans, the variants with higher p values were substantially more predictive of g ($r_{\text{high}_P} = 0.141$ vs. $r_{\text{low}_P} = 0.035$), while among European-Americans, this was not the case ($r_{\text{high}_P} = 0.265$ vs. $r_{\text{low}_P} = 0.263$). Cross-validation confirmed that the concordant PGS had higher predictive validity among the non-discovery population. However, the concordant PGS subset did not seem to have higher predictivity, based on cross-validation, than the combined set. The identical validity for concordant PGS could not be sustained in cross-validation. Thus, we do not use these disaggregated PGS in further analysis.

3.5. Path Analysis

Finally, we depict our results in path model form. As seen in Figure 4, substantial differences exist in the transethnic validity of the eduPGS. We therefore limited our path analysis to monoracial African-Americans. The model was run with the lavaan R package with missing data handled using listwise deletion. (Missing color scores were not MCAR, so we did not impute data.) Results are shown in Figure 5. We leave European ancestry unstandardized, as in previous analyses, while the other variables were standardized. Thus, the total effect of European ancestry on cognitive ability is calculated as (1) the unstandardized path coefficient between European ancestry and cognitive ability ($B = 0.512$) plus (2) the unstandardized path between European ancestry and color times the standardized path coefficient between color and cognitive ability ($-2.209 \times -0.066 = 0.146$) plus (3) the unstandardized path between European ancestry and eduPGS multiplied by the standardized path coefficient between eduPGS and cognitive ability ($2.515 \times 0.131 = 0.329$).

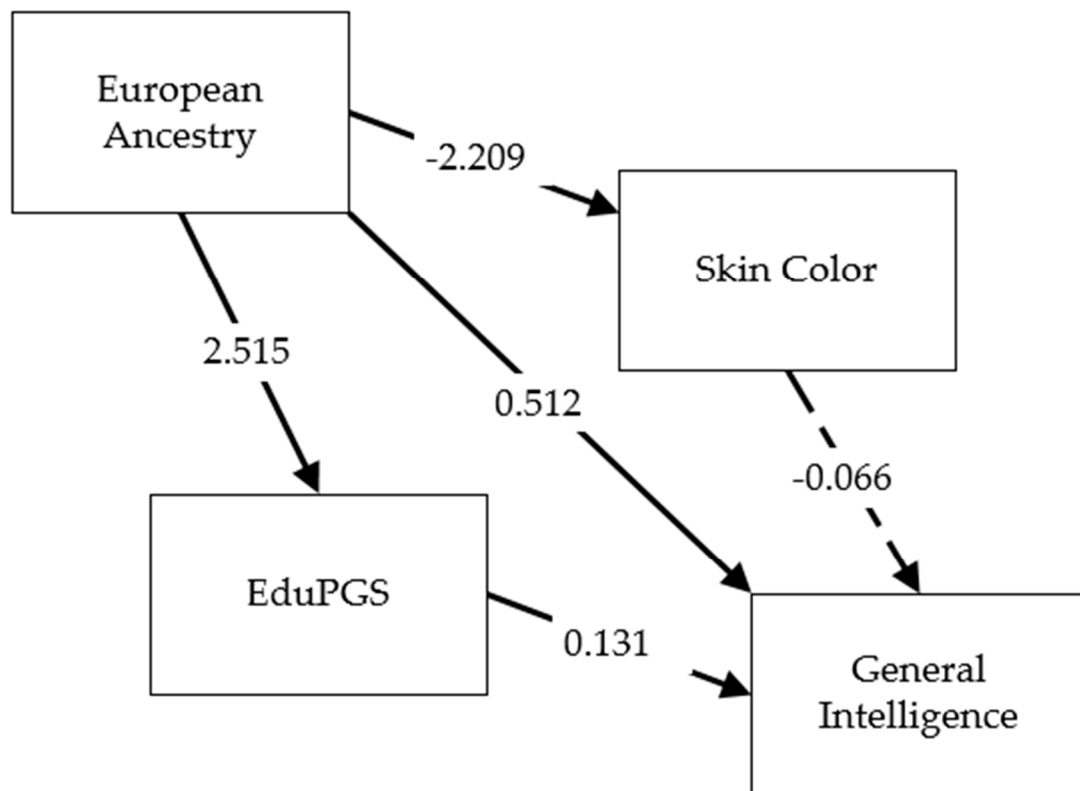


Figure 5. Path diagram for relation between European Ancestry, color, eduPGS, and g in the African-American sample.

Table 13 shows the estimates for the different paths. From the table, while European ancestry and eduPGS had significant effects, color did not (although it was trending toward significance, $p = 0.12$). Additionally, the covariance between color and eduPGS was not significant. The association between European ancestry and g is more substantially mediated by the cognition-related eduPGS than by color scores. An analysis of the effect of color within sibling pairs is included in the supplement but the results are extremely tentative due to a lack of statistical power and the unconfirmed validity of our measure of color.

Table 13. Detailed results for path diagram.

| | | | Unstandardized Estimate | S.E. | P value | Lower 95% CI | Upper 95% CI | Standardized Estimate |
|------------|---|------------|-------------------------|-------|---------|--------------|--------------|-----------------------|
| EUR | → | G | 0.512 | 0.255 | 0.045 | 0.013 | 1.012 | 0.059 |
| EUR | → | eduPGS | 2.515 | 0.156 | 0.000 | 2.209 | 2.821 | 0.381 |
| eduPGS | → | G | 0.131 | 0.036 | 0.000 | 0.060 | 0.202 | 0.099 |
| Skin Color | → | G | −0.066 | 0.043 | 0.120 | −0.150 | 0.017 | 0.043 |
| EUR | → | Skin Color | −2.209 | 0.133 | 0.000 | −2.469 | −1.949 | −0.392 |
| Skin Color | ~ | eduPGS | −0.020 | 0.012 | 0.089 | −0.044 | 0.003 | −0.044 |

Note: EUR = European ancestry.

3.6. The Spearman–Jensen Hypothesis

Spearman’s hypothesis holds that racial differences in cognitive ability are largely the result of differences in g. This theory predicts a strong positive correlation between subtest g loadings and the vector of group differences. We test this theory using Jensen’s method of correlated vectors (MCV; [2]) by examining the correlation between the vector of g loadings, the vector of subtest heritability, and four vectors of group differences. The vector of g loadings is the g loadings for the 10 subtests found to be MI for African and European-Americans. The vector of heritability is the average of the African-

and European-American heritabilities reported by Mollon et al. [65]. The vectors of group differences were: (1) the correlation with European ancestry (for the combined sample), (2) the correlation with MTAG 10K eduPGS for the European-American sample, (3) the correlation with MTAG 10k eduPGS for the monoracial African-American sample, and (4) the African-/European-American mean group differences. These results are shown in Table 14.

Table 14. Results from Jensen's Method of Correlated Vectors.

| | <i>g</i> Loading | B-W h^2 | Ancestry <i>r</i> | eduPGS <i>r</i> (European-American) | eduPGS <i>r</i> (African American) | African/European Gap |
|--|------------------|-----------|-------------------|--|---------------------------------------|-------------------------|
| <i>g</i> loading | 1 | | | | | |
| B-W h^2 | 0.372 | 1 | | | | |
| Ancestry <i>r</i> | 0.941 | 0.467 | 1 | | | |
| eduPGS <i>r</i> (European-American) | 0.892 | 0.604 | 0.993 | 1 | | |
| eduPGS <i>r</i> (African-American) | 0.901 | 0.600 | 0.836 | 0.846 | 1 | |
| African/European gap | 0.929 | 0.478 | 0.997 | 0.926 | 0.813 | 1 |

Note: *N* (subtest) = 10. eduPGS = education polygenic score, h^2 = heritability.

Spearman's hypothesis is strongly confirmed in our data. Not only is there a large Jensen effect on European ancestry, there is an equally large Jensen effect for the subtest correlations with the MTAG eduPGS. The correlations with the heritabilities are also positive, although these were estimated based on small samples (as far as behavior genetics goes) and thus this vector correlation is downwardly biased. If one includes the remaining 5 subtests, the correlations are about the same magnitude.

3.7. Assessment of Measure Invariance with Respect to Ancestry

Initially, we confirmed that MI held between self-identified African- and European-Americans. However, it is also possible to examine if MI holds with respect to genetic ancestry using LSEM (see [103]). A finding of MI along the whole range of admixture would preclude many group-specific factors which are commonly proposed to explain potential admixture-ability or other admixture-outcome relationships.

We fit an LSEM on the full African- and European-American sample ($n = 7399$) using 21 focal points (i.e., 5% ancestry increments ranging from 0% to 100%). From 0% to 100% European ancestry there was no deviation from MI. While the mean levels in *g* and complex reasoning increased linearly, the mean levels in episodic memory and executive functioning decreased linearly. MI appears to be tenable across the whole range of ancestry and, as well, the relationship between latent abilities and genetic ancestry comport with Spearman's hypothesis.

We additionally fitted LSEMs with SES. When standardized SES was modeled from its lowest to its greatest value, it showed measurement invariance but failed to account for the differences in the latent means between racial groups despite having a similar, albeit smaller, effect compared to European admixture when modeled as a background variable in the MGCFA model. The important takeaways from the LSEM results are that MI with respect to both SES and admixture is tenable, admixture can fully account for group mean differences in latent abilities while our present measure of SES cannot, and, as MI implies that the differences between groups on tests are due to differences in means and variances alone, this appears to be the case with admixture and race generally.

4. Discussion

We conducted an admixture study to assess the association between cognitive ability and European ancestry in the African- and European-American populations. Earlier researchers (e.g., [51,52]) were unable to detect significant relationships between ancestry and cognitive ability. We, however, found a strong, significant association between European genetic ancestry and cognitive ability, confirming the recent results of both Kirkegaard et al. [24] and Warne [25].

4.1. Global Admixture Analysis and Past Research Findings

The betas we calculated for European ancestry were very similar across models: $B = 0.78$ for the self-identified monoracial African-American-only sample (Model 1, Table 5) and $B = 0.75$ for the combined African- and European-American sample (with controls for SIRE; Model 2, Table 6). The former beta corresponds to a bivariate correlation of only $r = 0.086$ ($N = 2179$; Table 4). This small correlation is misleading as to its importance. The correlation is small in magnitude simply because the variation in European ancestry in the sample is low (or restricted). As Jensen [104] noted, constrained variability results in attenuated admixture correlations. Because of this, a small correlation within a population with restricted ancestry variance can be consistent with a large overall effect of admixture. For example, our African-American sample averaged 18.7% ($SD = 11.7\%$) European ancestry. Compared with European-Americans (98.6%), this is a difference of $(98.6\% - 18.7\%) / 11.7\% = 6.83$ standard deviations of European ancestry. Thus, the predicted difference in cognitive ability between African- and European-Americans (or, rather, African-Americans with the same amount of European ancestry as European-Americans in this sample), based on our observed bivariate association, would be $r = 0.086 \times 6.83 = 0.587$ SD. This is relative to the African-/European-American difference of $d = 0.984$ in cognitive ability (from Table 1). Thus, this small $r = 0.086$ correlation is consistent with 60% of the mean difference being explained by genetic ancestry.

Note that the correlation Scarr et al. [52] found was $0.05/0.086 = 58\%$ of the correlation we found. The attenuation in correlation is likely attributable to the reduced validity of their admixture index and the lower heritability of their measure of cognitive ability. Thus, our study largely vindicates Jensen's [104] reanalysis of the Scarr et al. [52] data.

We found very large Jensen effects for both the African-/European-American difference ($r = 0.929$) and the association between ancestry and g loadings ($r = 0.941$). The finding of a Jensen effect on ancestry is also consistent with the results presented by Scarr et al. [52]. As Loehlin [105] seems to have implied, the association between admixture and ability in the Scarr et al. [52] sample comported with the typical weak form of Spearman's hypothesis in that European ancestry was positively related ($r = 0.1$) with Raven's Progressive Matrices scores and negatively related ($r = -0.12$) with a memory measure. Indeed, using average g loadings provided by Jensen [19] (cf. [106]) the correlation between the African-/European-American score differences and g loadings is $r = 0.64$, while the correlation between the African-American-only g loadings and the score differences between the most- and least-African in this group is $r = 0.84$.

The only other study which has examined Spearman's hypothesis in the context of intra-SIRE ancestry-related differences is Hu et al. [18]. The authors studied participants from the National Longitudinal Survey of Youth 1997. Among African-Americans, Hu et al. [17]) found that darker color was associated with lower g scores while parent-reported European ancestry was associated with higher g scores. Jensen effects were found for both indices of ancestry. It seems that Spearman's hypothesis generalizes to ancestry-related differences within self-identified racial groups.

Our study goes beyond those of Kirkegaard et al. [24]) and Warne [25]) in that we (as with Hu et al. [17]) established both SFI and the weak form of Spearman's hypothesis regarding the African-/European-American difference. On the basis of this result we conclude that the SIRE differences in this battery are typically-sized and consistent with prior investigations of ability patterns in African- and European-American samples. Because the test battery is unbiased and it assesses the same traits in both populations, the difference observed here is a real difference in the cognitive abilities being measured.

We go beyond Hu et al. [17] in that we included a measure of both color and genetic ancestry in our regression analyses. As predicted by the human capital model (Hu et al. [17], Prediction 7), we found that color had no incremental validity beyond ancestry. This lack of incremental validity for color is possibly due to power. Color scores were available for only a subset of individuals since several of the arrays did not include all the SNPs needed by the algorithm to compute the scores. Thus, we cannot completely rule out an incremental effect of color in the present dataset. We can, however,

conclude that the association between European ancestry and g is not secondary to that between color and g .

It is worth noting that in the path model we did not find a significant association between skin color and eduPGS. This could be due to a lack of statistical power. This may, however, suggest a limited role for assortative mating (or, for that matter, pleiotropy), which has been suggested by some (e.g., [2]) in explaining the color-IQ association among African-Americans.

Beyond color, we also controlled for SES. Although not as strong of a predictor as ancestry, SES was a useful predictor of cognitive ability which modestly attenuated the effect of ancestry on cognitive ability. These results, however, are expected in both genetic and non-genetic models because environmental variables and familial characteristics like SES also reflect genetic influences [2,107,108]. Controlling for these variables may therefore spuriously weaken the overall association between ancestry and the relevant genetic factors that actually explain their influence. Conditions in which cross-sectional data reflect real mediation are exceptional [109] so future investigations should attempt to tease out causality with longitudinal designs and local admixture mapping. A particularly strong research design would be to investigate sibling variation in admixture just as Visscher et al. [110] did with relatedness. Very large samples are needed for this design if using full siblings; if data can be found using half- or adopted-siblings, then the required sample sizes would be more reasonable.

4.2. eduPGS Findings and Past Research

We also evaluated the transethnic validity of eduPGS. We found that the eduPGS with the highest validity for g in both the African- and European-American samples was the MTAG_EA_10K set. Although the validity in the African-American sample was approximately half of that in the European-American sample ($r_{AA} = 0.1115$; $r_{EA} = 0.2269$), the relations were statistically significant in both populations ($p < 0.0001$). As with Piffer [111] we found large African-/European-American differences in these eduPGS ($d = 1.89$). Using the beta in the African-American sample and controlling for the effect of European ancestry ($B = 0.124$; Model 1b; Table 10), we estimate that the known eduPGS can naïvely explain as much as 20%–25% of the African-/European-American intelligence gap.

Removing variants with low MAF in African 1000 Genomes lineages had little effect on the validity of MTAG_10K eduPGS among African and European-Americans. Thus, contrary to some arguments, European-specific alleles do not seem to be biasing prediction with eduPGS. Our results corroborate those of Piffer [111] who found a strong ecological correlation between MTAG-derived SNPs and population IQ ($r = 0.86$). We also found, using both regression and path analysis that, while the eduPGS mediates the association between European ancestry and cognitive ability, skin color scores do not.

4.3. Alternative Explanations and Methods to Further Reduce Uncertainty

At least one researcher has advanced the possibility that admixture designs are hopelessly confounded due to historical assortative mating [112]. This theory supposes that the European ancestors of African-Americans were genetically disposed toward either lower or higher cognitive ability in a systematic way. Since Piffer [111] found that other African descent groups have similarly low eduPGS, this explanation would necessitate that the eduPGS are not indexing genetic differences related to cognitive ability in African groups. Determining if this is the case is important for future research. We can directly investigate this hypothesis and further test the transethnic validity of the eduPGS with local admixture mapping. If the theory implied here is correct, European ancestry segments should have systematically higher or lower eduPGS in African-American samples. Relatedly, if eduPGS lack transethnic validity in Africans, they will not be associated with specific regions of the brain, biological variables, or consistent patterns of gene expression as they are in Europeans.

Our data are compatible with a between-group heritability (variance explained by European ancestry) of between 50% and 70% depending on the model chosen (see Scarr et al., p. 85 [52]). This estimate of between-group heritability is consistent with Rushton and Jensen's [9] hereditarian

model, according to which 50%–80% of the African-/European-American cognitive difference is due to genetic differences.

While the statistical mediation by PGS scores suggests that genetic factors may be involved, as discussed in detail by Kirkegaard et al. [24], we cannot rule out many types of confounding environmental variables with this research design. Global admixture analysis results are suggestive and should only be considered a first step for investigating the effects of admixture on a trait. We suggest to attempt replication of the current results using a nationally representative sample and then, if these findings are confirmed, proceed to admixture mapping (local admixture analysis). This is the standard approach taken in medical epidemiology.

We suggest two approaches to further reduce the uncertainty regarding the causes of the African-/European-American cognitive ability gap. First, attempts should be made to replicate the current results using other samples (e.g., the Add Health study; [64]). Second, local ancestry analysis/admixture mapping to examine the regions of the genome where the association with ancestry is most pronounced are a natural follow-on project. The rationale of such an analysis has been explained by others (e.g., [17,24,113,114]). It would also be worthwhile to attempt to replicate these results in admixed American populations outside of the United States (e.g., Brazil, Colombia, etc.). This research project can and should be expanded to other ethnic groups both in and outside of the United States. Examples include Aborigines in Australia, Cape Coloureds in South Africa, and the Mestizo population in Mexico. For some groups, it may be of utility to examine differences in broad abilities (e.g., spatial or mathematical ability) instead of general ability as done here, as differences may not be general.

4.4. General Conclusion

Rushton and Jensen [9] called for modern genetic studies to test the hereditarian model. They predicted that “for those Black individuals who possess more White genes, their physical, behavioral, and other characteristics will approach those of Whites” ([9], p. 262). In the present study, we confirmed that this was the case for general cognitive ability. Moreover, we showed that the association between European ancestry and *g* was substantially mediated by eduPGS rather than skin color PGS. These results provide support for a hereditarian model.

We conducted several analyses aimed at testing a genetic hypothesis for the African-/European-American difference in cognitive ability. We found that European ancestry was a consistent predictor of cognitive ability, even after entering various controls into our models. The large observed eduPGS differences were capable of predicting substantial proportions of the cognitive ability gap, which suggests the possibility of mediation. Future assessments with cross-racially valid PGS should attempt to assess this possibility more clearly. Our global admixture-based results suggest a contribution of admixture to the African-/European-American cognitive ability gap and should encourage future investigations at the level of local admixture.

Supplementary Materials: The following are available online at <http://www.mdpi.com/2624-8611/1/1/34/s1>.

Author Contributions: Conceptualization, J.L., J.F., E.K.; methodology, J.L., E.K., B.P., J.F.; software, J.L. and E.K.; validation, J.L. and E.K.; formal analysis, J.L., E.K., B.P., and J.F.; investigation, J.L. and E.K.; resources, J.F. and B.P.; data curation, J.F. and B.P.; writing—original draft preparation, J.L.; writing—review and editing, J.L., E.K. and J.F.; visualization, J.L.; supervision, B.P.

Funding: This research received no external funding.

Acknowledgments: Gur, Hakonarson, and collaborators request that publications resulting from these data cite their original publication: [TBD]. Support for the collection of the data sets was provided by grant RC2MH089983 awarded to Raquel Gur and RC2MH089924 awarded to Hakon Hakonarson. All subjects were recruited through the Center for Applied Genomics at The Children’s Hospital in Philadelphia. dbGaP accession phs000607.v1.p1.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. Shuey, A. *The testing of Negro Intelligence*; Social Science Press: New York, NY, USA, 1996.

2. Jensen, A.R. *The g Factor: The Science of Mental Ability*; Praeger Publishers/Greenwood Publishing Group: Westport, CT, USA, 1998.
3. Roth, P.L.; Bevier, C.A.; Bobko, P.; Switzer, F.S.; Tyler, P. ETHNIC GROUP DIFFERENCES IN COGNITIVE ABILITY IN EMPLOYMENT AND EDUCATIONAL SETTINGS: A META-ANALYSIS. *Pers. Psychol.* **2001**, *54*, 297–330. [[CrossRef](#)]
4. Pesta, B.P.; Kirkegaard, E.O.; te Nijenhuis, J.; Fuerst, G.R. Racial and Ethnic Group Differences in the Heritability of Intelligence: A Systematic Review and Meta-Analysis. 2019; Manuscript submitted for publication.
5. Sesardic, N. *Making Sense of heritability*; Cambridge University Press: Cambridge, UK, 2005.
6. Jensen, A.R. How much can we boost IQ and scholastic achievement? *Harv. Educ. Rev.* **1969**, *39*, 1–123. [[CrossRef](#)]
7. Jensen, A.R. *Educability and Group Differences*, 1st U.S. ed.; Harper & Row: New York, NY, USA, 1973.
8. Lynn, R. *Race Differences in Intelligence: An Evolutionary Analysis Second Revised Edition*; Washington Summit Books: Augusta, GA, USA, 2015.
9. Rushton, J.P.; Jensen, A.R. Thirty years of research on race differences in cognitive ability. *Psychol. Public Policy Law* **2005**, *11*, 235–294. [[CrossRef](#)]
10. Jensen, A.R. Reducing the heredity-environment uncertainty. *Harv. Educ. Rev.* **1969**, *39*, 449–483. [[CrossRef](#)]
11. Rowe, D.C. Under the skin: On the impartial treatment of genetic and environmental hypotheses of racial differences. *Am. Psychol.* **2005**, *60*, 60–70. [[CrossRef](#)] [[PubMed](#)]
12. Scarr, S.; Weinberg, R.A. Intellectual similarities within families of both adopted and biological children. *Intelligence* **1977**, *1*, 170–191. [[CrossRef](#)]
13. Weinberg, R.A.; Scarr, S.; Waldman, I.D. The Minnesota transracial adoption study: A follow-up of IQ test performance at adolescence. *Intelligence* **1992**, *16*, 117–135. [[CrossRef](#)]
14. Turkheimer, E. Individual and group differences in adoption studies of IQ. *Psychol. Bull.* **1991**, *110*, 392–405. [[CrossRef](#)]
15. Thomas, D. Racial IQ Differences among Transracial Adoptees: Fact or Artifact? *J. Intell.* **2017**, *5*, 1. [[CrossRef](#)] [[PubMed](#)]
16. Murray, C. The Secular Increase in IQ and Longitudinal Changes in the Magnitude of the Black-White Difference: Evidence from the NLSY. In Proceedings of the Behavior Genetics Association Meeting, Vancouver, BC, Canada, 4 July 1999.
17. Hu, M.; Lasker, J.; Kirkegaard, E.O.; Fuerst, J.G. Filling in the Gaps: The Association between Intelligence and Both Color and Parent-Reported Ancestry in the National Longitudinal Survey of Youth 1997. *Psych* **2019**, *1*, 240–261. [[CrossRef](#)]
18. Jensen, A.R. The nature of the black–white difference on various psychometric tests: Spearman’s hypothesis. *Behav. Brain Sci.* **1985**, *8*, 193–219. [[CrossRef](#)]
19. McDaniel, M.A.; Kepes, S. An Evaluation of Spearman’s Hypothesis by Manipulating g Saturation. *Int. J. Sel. Assess.* **2014**, *22*, 333–342. [[CrossRef](#)]
20. Nijenhuis, J.T.; Hoek, M.V.D.; Dragt, J. A Meta-Analysis of Spearman’s Hypothesis Tested on Latin-American Hispanics, including a New Way to Correct for Imperfectly Measuring the Construct of g. *Psych* **2019**, *1*, 101–122. [[CrossRef](#)]
21. Rowe, D.C.; Cleveland, H.H. Academic achievement in Blacks and Whites: Are the developmental processes similar? *Intelligence* **1996**, *23*, 205–228. [[CrossRef](#)]
22. Loehlin, J.C.; Lindzey, G.; Spuhler, J.N. Cross-group Comparisons of Intellectual Abilities. In *Race Differences in Intelligence*; Freeman: San Francisco, CA, USA, 1975; pp. 170–171.
23. Shockley, W. Possible transfer of metallurgical and astronomical approaches to problem of environment versus ethnic heredity. *Science* **1966**, *154*, 148.
24. Kirkegaard, E.O.W.; Menie, M.W.O.; Williams, R.L.; Fuerst, J.; Meisenberg, G. Biogeographic Ancestry, Cognitive Ability and Socioeconomic Outcomes. *Psych* **2019**, *1*, 1–25. [[CrossRef](#)]
25. Warne, R.T. Genetic Ancestry and Cognitive Ability. 2019; Manuscript submitted for publication.
26. Templeton, A.R. The Genetic and Evolutionary Significance of Human Races. In *Race and Intelligence: Separating Science from Myth*; Fish, J.M., Ed.; Routledge: Abingdon, UK, 2001.
27. Reed, T.E. “The genetic hypothesis”: It was not tested but it could have been. *Am. Psychol.* **1997**, *52*, 77–78. [[CrossRef](#)]

28. Rushton, J.P.; Jensen, A.R. Race and IQ: A Theory-Based Review of the Research in Richard Nisbett's Intelligence and How to Get It. *Open Psychol. J.* **2010**, *3*, 9–35. [[CrossRef](#)]
29. Bouchard, T.J. The Wilson Effect: The increase in heritability of IQ with age. *Twin Res. Hum. Genet.* **2013**, *16*, 923–930. [[CrossRef](#)]
30. Bouchard, T.J.; McGue, M. Genetic and environmental influences on human psychological differences. *J. Neurobiol.* **2003**, *54*, 4–45. [[CrossRef](#)]
31. Briley, D.A.; Tucker-Drob, E.M. Explaining the Increasing Heritability of Cognitive Ability Across Development: A Meta-Analysis of Longitudinal Twin and Adoption Studies. *Psychol. Sci.* **2013**, *24*, 1704–1713. [[CrossRef](#)] [[PubMed](#)]
32. Becker, N.S.; Verdu, P.; Froment, A.; Le Bomin, S.; Pagezy, H.; Bahuchet, S.; Heyer, E. Indirect evidence for the genetic determination of short stature in African Pygmies. *Am. J. Phys. Anthr.* **2011**, *145*, 390–401. [[CrossRef](#)] [[PubMed](#)]
33. Jarvis, J.P.; Scheinfeldt, L.B.; Soi, S.; Lambert, C.; Omberg, L.; Ferwerda, B.; Froment, A.; Bodo, J.-M.; Beggs, W.; Hoffman, G.; et al. Patterns of Ancestry, Signatures of Natural Selection, and Genetic Association with Stature in Western African Pygmies. *PLoS Genet.* **2012**, *8*, e1002641. [[CrossRef](#)] [[PubMed](#)]
34. Romero, I.G.; Leavesley, M.; Sherman, B.T.; Siddie, K.; Kaitokai, E.; Babalu, A.; Belatti, M.; Hopkinshaw, B.; Shaw, C.; Nelis, M.; et al. Evolution of the Pygmy Phenotype: Evidence of Positive Selection from Genome-wide Scans in African, Asian, and Melanesian Pygmies. *Hum. Biol.* **2013**, *85*, 251.
35. Pemberton, T.J.; Verdu, P.; Becker, N.S.; Willer, C.J.; Hewlett, B.S.; Le Bomin, S.; Froment, A.; Rosenberg, N.A.; Heyer, E. A genome scan for genes underlying adult body size differences between Central African hunter-gatherers and farmers. *Hum. Genet.* **2018**, *137*, 487–509. [[CrossRef](#)]
36. Perry, G.H.; Foll, M.; Grenier, J.-C.; Patin, E.; Nedelec, Y.; Pacis, A.; Barakatt, M.; Gravel, S.; Zhou, X.; Nsobya, S.L.; et al. Adaptive, convergent origins of the pygmy phenotype in African rainforest hunter-gatherers. *Proc. Natl. Acad. Sci. USA* **2014**, *111*, E3596–E3603. [[CrossRef](#)] [[PubMed](#)]
37. Cheng, C.-Y.; Reich, D.; Haiman, C.A.; Tandon, A.; Patterson, N.; Elizabeth, S.; Akyzbekova, E.L.; Brancati, F.L.; Coresh, J.; Boerwinkle, E.; et al. African Ancestry and Its Correlation to Type 2 Diabetes in African Americans: A Genetic Admixture Analysis in Three U.S. Population Cohorts. *PLoS ONE* **2012**, *7*, e32840. [[CrossRef](#)]
38. Williams, R.C.; Long, J.C.; Hanson, R.L.; Sievers, M.L.; Knowler, W.C. Individual Estimates of European Genetic Admixture Associated with Lower Body-Mass Index, Plasma Glucose, and Prevalence of Type 2 Diabetes in Pima Indians. *Am. J. Hum. Genet.* **2000**, *66*, 527–538. [[CrossRef](#)]
39. Florez, J.C.; Price, A.L.; Campbell, D.; Riba, L.; Parra, M.V.; Yu, F.; Duque, C.; Saxena, R.; Gallego, N.; Tello-Ruiz, M.; et al. Strong association of socioeconomic status with genetic ancestry in Latinos: Implications for admixture studies of type 2 diabetes. *Diabetologia* **2009**, *52*, 1528–1536. [[CrossRef](#)]
40. Kosoy, R.; Qi, L.; Nassir, R.; Garcia, L.; Allison, M.; Shigeta, R.; Seldin, M.F. Relationship between hypertension and admixture in post-menopausal African American and Hispanic American women. *J. Hum. Hypertens.* **2012**, *26*, 365–373. [[CrossRef](#)]
41. Jeong, C.; Alkorta-Aranburu, G.; Basnyat, B.; Neupane, M.; Witonsky, D.B.; Pritchard, J.K.; Beall, C.M.; Di Rienzo, A. Admixture facilitates genetic adaptations to high altitude in Tibet. *Nat. Commun.* **2014**, *5*, 3281. [[CrossRef](#)] [[PubMed](#)]
42. Flores, C.; Ma, S.-F.; Pino-Yanes, M.; Wade, M.S.; Pérez-Méndez, L.; Kittles, R.A.; Wang, D.; Papaiahgari, S.; Ford, J.G.; Kumar, R.; et al. African Ancestry Is Associated with Asthma Risk in African Americans. *PLoS ONE* **2012**, *7*, e26807. [[CrossRef](#)] [[PubMed](#)]
43. Halder, I.; Matthews, K.A.; Buysse, D.J.; Strollo, P.J.; Causser, V.; Reis, S.E.; Hall, M.H. African Genetic Ancestry is Associated with Sleep Depth in Older African Americans. *Sleep* **2015**, *38*, 1185–1193. [[CrossRef](#)]
44. Freedman, M.L.; Haiman, C.A.; Patterson, N.; McDonald, G.J.; Tandon, A.; Waliszewska, A.; Penney, K.; Steen, R.G.; Ardlie, K.; John, E.M.; et al. Admixture mapping identifies 8q24 as a prostate cancer risk locus in African-American men. *Proc. Natl. Acad. Sci. USA* **2006**, *103*, 14068–14073. [[CrossRef](#)]
45. Gudmundsson, J.; Sulem, P.; Manolescu, A.; Amundadottir, L.T.; Gudbjartsson, D.; Helgason, A.; Rafnar, T.; Bergthorsson, J.T.; Agnarsson, B.A.; Baker, A.; et al. Genome-wide association study identifies a second prostate cancer susceptibility variant at 8q24. *Nat. Genet.* **2007**, *39*, 631–637. [[CrossRef](#)] [[PubMed](#)]
46. Yeager, M.; Orr, N.; Hayes, R.B.; Jacobs, K.B.; Kraft, P.; Wacholder, S.; Minichiello, M.J.; Fearnhead, P.; Yu, K.; Chatterjee, N.; et al. Genome-wide association study of prostate cancer identifies a second risk locus at 8q24. *Nat. Genet.* **2007**, *39*, 645–649. [[CrossRef](#)]

47. Han, Y.; Rand, K.A.; Hazelett, D.J.; Ingles, S.A.; Kittles, R.A.; Strom, S.S.; Rybicki, B.A.; Nemesure, B.; Isaacs, W.B.; Stanford, J.L.; et al. Prostate Cancer Susceptibility in Men of African Ancestry at 8q24. *J. Natl. Cancer Inst.* **2016**, *108*, 431. [[CrossRef](#)]
48. Giri, A.; Edwards, T.L.; Hartmann, K.E.; Torstenson, E.S.; Wellons, M.; Schreiner, P.J.; Edwards, D.R.V. African genetic ancestry interacts with body mass index to modify risk for uterine fibroids. *PLoS Genet.* **2017**, *13*, e1006871. [[CrossRef](#)] [[PubMed](#)]
49. Paiva, S.G. Fatores de Risco Para Doenças Cardiovasculares em Quilombos Contemporâneos do Brasil Central: Parâmetros Demográficos, Socioeconômicos, Ancestralidade Genética e Saúde. Ph.D. Thesis, University of Brasília, Brasília, Brazil, 2017.
50. Pearl, J. *Causality: Models, Reasoning and Inference*, 2nd ed.; Cambridge University Press: Cambridge, UK, 2012.
51. Loehlin, J.C.; Vandenberg, S.G.; Osborne, R.T. Blood group genes and negro-white ability differences. *Behav. Genet.* **1973**, *3*, 263–270. [[CrossRef](#)]
52. Scarr, S.; Pakstis, A.J.; Katz, S.H.; Barker, W.B. Absence of a relationship between degree of white ancestry and intellectual skills within a black population. *Hum. Genet.* **1997**, *39*, 69–86. [[CrossRef](#)] [[PubMed](#)]
53. Rothhammer, F.; Llop, E. Amerindian descent and intellectual performance in Chilean university students. *Hum. Biol.* **1976**, *48*, 455–464. [[PubMed](#)]
54. Valenzuela, C.Y.; Pastene, C.S.; Pérez, C.M. Intelligence and genetic markers in Chilean children. *Biol. Res.* **1998**, *31*, 81–92. [[PubMed](#)]
55. Lima-Costa, M.F.; Macinko, J.; Mambrini, J.V.D.M.; Peixoto, S.V.; Pereira, A.C.; Tarazona-Santos, E.; Ribeiro, A.L.P. Genomic African and Native American Ancestry and Chagas Disease: The Bambui (Brazil) Epigen Cohort Study of Aging. *PLoS Negl. Trop. Dis.* **2016**, *10*, e0004724. [[CrossRef](#)] [[PubMed](#)]
56. Ruiz-Linares, A.; Adhikari, K.; Acuña-Alonzo, V.; Quinto-Sánchez, M.; Jaramillo, C.; Arias, W.; Fuentes, M.; Pizarro, M.; Everardo, P.; De Avila, F.; et al. Admixture in Latin America: Geographic Structure, Phenotypic Diversity and Self-Perception of Ancestry Based on 7342 Individuals. *PLoS Genet.* **2014**, *10*, e1004572. [[CrossRef](#)] [[PubMed](#)]
57. Conley, D.; Fletcher, J. *The Genome Factor: What the Social Genomics Revolution Reveals about Ourselves, Our History, and the Future*; Princeton University Press: Princeton, NJ, USA, 2017.
58. Condon, D.M.; Revelle, W. The international cognitive ability resource: Development and initial validation of a public-domain measure. *Intelligence* **2014**, *43*, 52–64. [[CrossRef](#)]
59. Bryc, K.; Durand, E.Y.; MacPherson, J.M.; Reich, D.; Mountain, J.L. The Genetic Ancestry of African Americans, Latinos, and European Americans across the United States. *Am. J. Hum. Genet.* **2015**, *96*, 37–53. [[CrossRef](#)] [[PubMed](#)]
60. Adhikari, K.; Chacón-Duque, J.C.; Mendoza-Revilla, J.; Fuentes-Guajardo, M.; Ruiz-Linares, A. The Genetic Diversity of the Americas. *Annu. Rev. Genom. Hum. Genet.* **2017**, *18*, 277–296. [[CrossRef](#)] [[PubMed](#)]
61. Halder, I.; Shriver, M.; Thomas, M.; Fernandez, J.R.; Frudakis, T. A panel of ancestry informative markers for estimating individual biogeographical ancestry and admixture from four continents: Utility and applications. *Hum. Mutat.* **2008**, *29*, 648–658. [[CrossRef](#)] [[PubMed](#)]
62. McQueen, M.B.; Boardman, J.D.; Domingue, B.W.; Smolen, A.; Tabor, J.; Killea-Jones, L.; Mullan Harris, K. The National Longitudinal Study of Adolescent to Adult Health (Add Health) Sibling Pairs Genome-Wide Data. *Behav. Genet.* **2015**, *45*, 12–23. [[CrossRef](#)] [[PubMed](#)]
63. Nisbett, R.E. *Intelligence and How to Get It: Why Schools and Cultures Count*; WW Norton & Company: New York, NY, USA, 2009.
64. Dolan, C.V. Investigating Spearman's Hypothesis by Means of Multi-Group Confirmatory Factor Analysis. *Multivar. Behav. Res.* **2000**, *35*, 21–50. [[CrossRef](#)] [[PubMed](#)]
65. Mollon, J.; Knowles, E.E.M.; Mathias, S.R.; Gur, R.; Peralta, J.M.; Weiner, D.J.; Robinson, E.B.; Gur, R.E.; Blangero, J.; Almasy, L.; et al. Genetic influence on cognitive development between childhood and adulthood. *Mol. Psychiatry* **2018**, *1*, 1. [[CrossRef](#)]
66. Calkins, M.E.; Merikangas, K.R.; Moore, T.M.; Burstein, M.; Behr, M.A.; Satterthwaite, T.D.; Ruparel, K.; Wolf, D.H.; Roalf, D.R.; Mentch, F.D.; et al. The Philadelphia Neurodevelopmental Cohort: Constructing a deep phenotyping collaborative. *J. Child Psychol. Psychiatry* **2015**, *56*, 1356–1369. [[CrossRef](#)]

67. Satterthwaite, T.D.; Connolly, J.J.; Ruparel, K.; Calkins, M.E.; Jackson, C.; Elliott, M.A.; Roalf, D.R.; Hopson, R.; Prabhakaran, K.; Behr, M.; et al. The Philadelphia Neurodevelopmental Cohort: A publicly available resource for the study of normal and abnormal brain development in youth. *NeuroImage* **2016**, *124*, 1115–1119. [CrossRef] [PubMed]
68. Moore, T.M.; Reise, S.P.; Gur, R.E.; Hakonarson, H.; Gur, R.C. Psychometric Properties of the Penn Computerized Neurocognitive Battery. *Neuropsychology* **2015**, *29*, 235–246. [CrossRef] [PubMed]
69. Templ, M.; Kowarik, A.; Alfons, A.; Prantner, B. VIM: Visualization and Imputation of Missing Values (Version 4.8.0). 2019. Available online: <https://CRAN.R-project.org/package=VIM> (accessed on 1 January 2019).
70. Templ, M.; Kowarik, A.; Filzmoser, P. Iterative stepwise regression imputation using standard and robust methods. *Comput. Stat. Data Anal.* **2011**, *55*, 2793–2806. [CrossRef]
71. Dolan, C.V.; Hamaker, E.L. Investigating Black-White differences in psychometric IQ: Multi-group confirmatory factor analyses of the WISC-R and K-ABC and a critique of the method of correlated vectors. In *Advances in Psychology Research*; Nova Science Publishers: Hauppauge, NY, USA, 2001; Volume 6, pp. 31–59.
72. Dolan, C.V.; Molenaar, P.C. Testing Specific Hypotheses Concerning Latent Group Differences in Multi-group Covariance Structure Analysis with Structured Means. *Multivar. Behav. Res.* **1994**, *29*, 203–222. [CrossRef]
73. Frisby, C.L.; Beaujean, A.A. Testing Spearman's hypotheses using a bi-factor model with WAIS-IV/WMS-IV standardization data. *Intelligence* **2015**, *51*, 79–97. [CrossRef]
74. Vyas, S.; Kumaranayake, L. Constructing socio-economic status indices: How to use principal components analysis. *Health Policy Plan.* **2006**, *21*, 459–468. [CrossRef]
75. Rindermann, H.; Ceci, S.J. Parents' Education Is More Important Than Their Wealth in Shaping Their Children's Intelligence: Results of 19 Samples in Seven Countries at Different Developmental Levels. *J. Educ. Gift.* **2018**, *41*, 298–326. [CrossRef]
76. Satterthwaite, T.D.; Elliott, M.A.; Ruparel, K.; Loughead, J.; Prabhakaran, K.; Calkins, M.E.; Mentch, F.D. Neuroimaging of the Philadelphia neurodevelopmental cohort. *Neuroimage* **2014**, *86*, 544–553. [CrossRef]
77. Chang, C.C.; Chow, C.C.; Tellier, L.C.; Vattikuti, S.; Purcell, S.M.; Lee, J.J. Second-generation PLINK: Rising to the challenge of larger and richer datasets. *GigaScience* **2015**, *4*, 7. [CrossRef] [PubMed]
78. Alexander, D.H.; Novembre, J.; Lange, K. Fast model-based estimation of ancestry in unrelated individuals. *Genome Res.* **2009**, *19*, 1655–1664. [CrossRef] [PubMed]
79. Chaitanya, L.; Breslin, K.; Zuniga, S.; Wirken, L.; Pośpiech, E.; Kukla-Bartoszek, M.; Sijen, T.; De Knijff, P.; Liu, F.; Branicki, W.; et al. The HIRISplex-S system for eye, hair and skin colour prediction from DNA: Introduction and forensic developmental validation. *Forensic Sci. Int. Genet.* **2018**, *35*, 123–135. [CrossRef] [PubMed]
80. Dixon, A.R.; Telles, E.E. Skin Color and Colorism: Global Research, Concepts, and Measurement. *Annu. Rev. Sociol.* **2017**, *43*, 405–424. [CrossRef]
81. Marira, T.D.; Mitra, P. Colorism: Ubiquitous Yet Understudied. *Ind. Organ. Psychol.* **2013**, *6*, 103–107. [CrossRef]
82. Parra, E.J.; A Kittles, R.; Shriver, M.D.; Shriver, M. Implications of correlations between skin color and genetic ancestry for biomedical research. *Nat. Genet.* **2004**, *36*, S54–S60. [CrossRef]
83. Leite, T.K.M.; Fonseca, R.M.C.; De França, N.M.; Parra, E.J.; Pereira, R.W. Genomic Ancestry, Self-Reported "Color" and Quantitative Measures of Skin Pigmentation in Brazilian Admixed Siblings. *PLoS ONE* **2011**, *6*, e27162. [CrossRef] [PubMed]
84. Lee, J.J.; Wedow, R.; Okbay, A.; Kong, E.; Maghziyan, O.; Zacher, M.; Nguyen-Viet, T.A.; Bowers, P.; Sidorenko, J.; Linnér, R.K.; et al. Gene discovery and polygenic prediction from a 1.1-million-person GWAS of educational attainment. *Nat. Genet.* **2018**, *50*, 1112–1121. [CrossRef]
85. Rosenberg, N.A.; Edge, M.D.; Pritchard, J.K.; Feldman, M.W. Interpreting polygenic scores, polygenic adaptation, and human phenotypic differences. *Evol. Med. Public Health* **2018**, *2019*, 26–34. [CrossRef]
86. Zanetti, D.; Weale, M.E. Transethnic differences in GWAS signals: A simulation study. *Ann. Hum. Genet.* **2018**, *82*, 280–286. [CrossRef]
87. Duncan, L.; Shen, H.; Gelaye, B.; Ressler, K.; Feldman, M.; Peterson, R.; Domingue, B. Analysis of polygenic score usage and performance across diverse human populations. *BioRxiv* **2018**, 398396. [CrossRef]
88. Spencer, A.V.; Cox, A.; Walters, K. Comparing the efficacy of SNP filtering methods for identifying a single causal SNP in a known association region. *Ann. Hum. Genet.* **2014**, *78*, 50–61. [CrossRef] [PubMed]

89. Wang, X.; Teo, Y.-Y. Trans-ethnic fine-mapping of rare causal variants. In *Assessing Rare Variation in Complex Traits*; Springer: New York, NY, USA, 2015; pp. 253–261.
90. Piffer, D. The European-African difference in Cognitive-Educational polygenic scores is moderated by the trans-ethnic validity of the SNPs. (under review).
91. Sirin, S.R. Socioeconomic Status and Academic Achievement: A Meta-Analytic Review of Research. *Rev. Educ. Res.* **2005**, *75*, 417–453. [[CrossRef](#)]
92. Hunter, J.E.; Schmidt, F.L. *Methods of Meta-Analysis: Correcting Error and Bias in Research Findings*; Sage: Thousand Oaks, CA, USA, 2004.
93. Walsh, S.; Chaitanya, L.; Clarisse, L.; Wirken, L.; Draus-Barini, J.; Kovatsi, L.; Maeda, H.; Ishikawa, T.; Sijen, T.; De Knijff, P.; et al. Developmental validation of the HIrisPlex system: DNA-based eye and hair colour prediction for forensic and anthropological usage. *Forensic Sci. Int. Genet.* **2014**, *9*, 150–161. [[CrossRef](#)] [[PubMed](#)]
94. Lynn, R. Skin Color and Intelligence in African Americans. *Popul. Environ.* **2002**, *23*, 365–375. [[CrossRef](#)]
95. Fuerst, J.G.R.; Lynn, R.; Kirkegaard, E.O.W. The Effect of Biracial Status and Color on Crystallized Intelligence in the U.S.-Born African-European American Population. *Psych* **2019**, *1*, 44–54. [[CrossRef](#)]
96. Kirkegaard, E.O.W.; Fuerst, J. Admixture in Argentina. *Mank. Q.* **2017**, *57*, 542–580.
97. Woodley, M.A.; Younuskunju, S.; Balan, B.; Piffer, D. Holocene Selection for Variants Associated with General Cognitive Ability: Comparing Ancient and Modern Genomes. *Twin Res. Hum. Genet.* **2017**, *20*, 271–280. [[CrossRef](#)]
98. Uricchio, L.H.; Kitano, H.C.; Gusev, A.; Zaitlen, N.A. An evolutionary compass for detecting signals of polygenic selection and mutational bias. *Evol. Lett.* **2019**, *3*, 69–79. [[CrossRef](#)]
99. Kim, M.S.; Patel, K.P.; Teng, A.K.; Berens, A.J.; Lachance, J. Genetic disease risks can be misestimated across global populations. *Genome Biol.* **2018**, *19*, 179. [[CrossRef](#)]
100. Amer, D.; Sirota, L. Beware the chopsticks gene. *Mol. Psychiatry* **2000**, *5*, 11–13.
101. Thompson, J. Piffer Kicks Against the Pricks. Available online: <http://www.unz.com/jthompson/piffer-kicks-against-the-pricks/> (accessed on 2 May 2019).
102. Marigorta, U.M.; Navarro, A. High Trans-ethnic Replicability of GWAS Results Implies Common Causal Variants. *PLoS Genet.* **2013**, *9*, e1003566. [[CrossRef](#)] [[PubMed](#)]
103. Hildebrandt, A.; Lüdtke, O.; Robitzsch, A.; Sommer, C.; Wilhelm, O. Exploring Factor Model Parameters across Continuous Variables with Local Structural Equation Models. *Multivar. Behav. Res.* **2016**, *51*, 257–258. [[CrossRef](#)] [[PubMed](#)]
104. Jensen, A.R. Obstacles, Problems, and Pitfalls in Differential Psychology. In *Race, Social Class, and Individual Differences in IQ*; Erlbaum: Hillsdale, NJ, USA, 1981.
105. Loehlin, J.C. Group differences in intelligence. In *Handbook of Intelligence*; Cambridge University Press: New York, NY, USA, 2000; pp. 176–193.
106. Scarr, S. *Race, Social Class, and Individual Differences in IQ*; Erlbaum: Hillsdale, NJ, USA, 1981.
107. Plomin, R.; Bergeman, C.S. The nature of nurture: Genetic influence on “environmental” measures. *Behav. Brain Sci.* **1991**, *14*, 373–386. [[CrossRef](#)]
108. Vinkhuyzen, A.A.E.; Van Der Sluis, S.; De Geus, E.J.C.; Boomsma, D.I.; Posthuma, D. Genetic influences on ‘environmental’ factors. *Genes Brain Behav.* **2010**, *9*, 276–287. [[CrossRef](#)]
109. Cole, D.A.; Maxwell, S.E. Testing Mediational Models with Longitudinal Data: Questions and Tips in the Use of Structural Equation Modeling. *J. Abnorm. Psychol.* **2003**, *112*, 558–577. [[CrossRef](#)] [[PubMed](#)]
110. Visscher, P.M.; Medland, S.E.; Ferreira, M.A.R.; Morley, K.I.; Zhu, G.; Cornes, B.K.; Montgomery, G.W.; Martin, N.G. Assumption-Free Estimation of Heritability from Genome-Wide Identity-by-Descent Sharing between Full Siblings. *PLoS Genet.* **2006**, *2*, e41. [[CrossRef](#)] [[PubMed](#)]
111. Piffer, D. Evidence for Recent Polygenic Selection on Educational Attainment and Intelligence Inferred from Gwas Hits: A Replication of Previous Findings Using Recent Data. *Psych* **2019**, *1*, 55–75. [[CrossRef](#)]
112. Centerwall, B.S. The use of racial admixture as evidence in intelligence research: A critique. *Hum. Genet.* **1978**, *45*, 237–238. [[CrossRef](#)]

113. Shriner, D. Overview of admixture mapping. *Curr. Protoc. Hum. Genet.* **2013**, *76*, 1–23. [[CrossRef](#)]
114. Winkler, C.A.; Nelson, G.W.; Smith, M.W. Admixture mapping comes of age. *Annu. Rev. Genom. Hum. Genet.* **2010**, *11*, 65–89. [[CrossRef](#)] [[PubMed](#)]



© 2019 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<http://creativecommons.org/licenses/by/4.0/>).